JNP

Ethylene Glycol Poisoning: Resolution of Cranial Nerve Deficit

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ABSTRACT

Ethylene glycol poisoning is a major contributor to the development of idiopathic metabolic acidosis, which may lead to renal failure. Ethylene glycol poisoning should be among suspected differentials when assessing a seemingly intoxicated patient with hypocalcemia, anion gap acidosis, and nontoxic blood alcohol levels. Glycol intoxication may lead to delayed neurologic manifestations, which may lead to an inaccurate diagnosis. No clear clinical guidelines exist to recommend treatment for this late effect. This is a case of the complete resolution of facial nerve deficit secondary to glycol poisoning after the completion of a steroid therapy course on an inpatient psychiatric unit.

Keywords: calcium oxalate crystals, cranial nerve deficit, ethylene glycol, fomepizole, ingestion, metabolic acidosis, neurological sequelae of glycol intoxication, palsy, poisoning, suicide, toxicity

Nurse practitioners (NPs) are increasingly on the front lines of primary care and imbedded within specialty care areas to address the vast array of undifferentiated complaints, including patients who have ingested poison. Ethylene glycol (EG) is the primary component in most automotive antifreeze solutions. In 2009, according to the American Association of Poison Control Centers, 5,282 exposures to EG were reported.[1] EG is known to taste sweet, and for this reason many animals and children are drawn to this toxin.

CASE REPORT

This is the case of a 53-year-old white man who was brought to his local emergency department (ED) secondary to an acute change in his mental status consistent with alcohol intoxication. His wife reported that over the past 24 hours he had become progressively incoherent and agitated.

In the ED, he was found to be in severe acidosis (arterial blood gas: pH = 7.00, pCO_2 = 19.9, pO_2 = 311, HCO_3 = 4.9, base excess = −24.8, O_2 saturation = 99.7). He was also found to be in acute renal failure and hyperkalemic (serum calcium = 8.3, serum potassium = 6.4, creatinine = 2.47, anion gap = 28). The patient was chemically sedated, intubated, and placed on mechanical ventilation for airway management. A sodium bicarbonate drip was used to correct his metabolic acidosis, and he was treated for hyperkalemia with emergent dialysis. The patient was also started on fomepizole 15 mg/kg and admitted to the medical intensive care unit. Once the patient was medically stable and breathing without support, he reported ingesting antifreeze to commit suicide. The patient's psychiatric needs were addressed by transferring him to the community hospital's inpatient psychiatric unit. Three days later at the request of his wife, the patient was transferred to another inpatient psychiatric unit of a large academic medical center.

In the academic medical center, the patient was admitted to the inpatient psychiatric service and was determined to have major depression. The NP service assumed care of the patient's ongoing medical needs while his mental health needs were addressed in the inpatient psychiatric unit.

Cranial Nerve Deficit

On day 17 after EG ingestion, the patient developed a left-sided facial droop and slurred speech and lost the ability to close his left eye. The patient also complained of sensation of facial heaviness and numbness and was aware that he was drooling. Staff members were not alerted until 2 hours after the onset of symptoms (after he had finished breakfast

and showered) because the patient was embarrassed by his condition. A rapid neurologic assessment was performed by the hospital stroke team. The patient underwent a computed tomographic scan of the head without contrast. There was no evidence of acute infarction, hemorrhage, mass, or lesion. Subsequently, magnetic resonance imaging (MRI) of the brain without contrast further confirmed the negative head computed tomographic scan findings. Neurology signed off on the case.

The next day he was seen by the NP and was noted to have symmetrical strength bilaterally and equal function of both upper and lower extremities. The physical examination remained consistent for left-side facial droop, asymmetrical forehead creases, and incomplete left eye closure. The examination remained negative for lateralizing weakness and pronator drift. The clinical presentation and history were discussed with a radiology resident who recommended another MRI of the head without contrast with the internal auditory canal (IAC) protocol.

MRI With IAC Protocol

The IAC protocol is usually performed for the following indications: evaluation of a cerebellopontine angle mass, inner ear dysfunction, or pathology of cranial nerves VII and VIII (as was the case in patients suffering from glycol poisoning). There are 5 main differences between the IAC protocol and standard brain MRI:

1. The IAC protocol focuses on the axial and coronal T1 sequences only through the IAC rather than the whole brain
2. A fast imaging employing steady-state acquisition sequence through the IACs is obtained axially and reformatted in the coronal plane (3-dimensional fast imaging employing steady-state acquisition sequence allows high-resolution reformatting in any plane)
3. Postcontrast images are limited to the IACs (but continued through to the parotid gland when intending to image for cranial nerve pathology)
4. The IAC protocol uses fat suppression for the postcontrast images because the region of interest (IAC) has more fat surrounding it than most brain parenchyma (which has very little fat surrounding it)
5. The IAC protocol does not perform a gradient echo sequence, which is more sensitive for bleeding

MRI with the IAC protocol showed enhancement of multiple cranial nerves, most prominently cranial nerve V and VII. MRI revealed enhancement of cranial nerves IX, X, and XII and enhancement of the right cochlear nerve. These radiologic findings may be seen in the setting of EG toxicity.[2-5]

Treatment of Cranial Nerve Deficit

The patient was started on a course of prednisone 60 mg by mouth, daily for 7 days, followed by a 10 mg/d taper for 7 days (totaling 14 days). The patient continued to receive hemodialysis 3 times per week for the duration of his 41-day stay. Seven days after the completion of the steroid taper (48 days after ingestion), the facial droop completely resolved, and the patient was able to close his left eye. It is not clear as to why the steroid course was efficacious, but speculation suggests an anti-inflammatory effect of the corticosteroid on the cranial nerves because postmortem examinations indicated inflammation because of oxalate crystal deposit. This isolated case suggests that the effect of corticosteroids on the resolution of cranial nerve palsy was remarkable. Further research is needed to substantiate this as a definitive treatment option.

PATHOPHYSIOLOGY

The toxic effects of EG are the result of the central nervous system depressant effects, which are similar to the effects of more commonly consumed ethyl alcohol. In part, EG is metabolized thorough the cytochrome P450 pathway by the enzyme alcohol dehydrogenase as it passes through the gastrointestinal tract and by the enzyme mixed function oxidase system in the liver.[6] EG is readily absorbed by the gastrointestinal tract and easily redistributed throughout the body while producing toxic metabolites that can remain for several days. The toxic effects of EG become more pronounced once the toxin is metabolized by these 2 processes. One of the metabolites of EG is glycolic acid, which is thought to be responsible for the severe acidosis that is found in patients with EG intoxication. Glycolic