Formic acid, when combined with sulfuric acid, gets dehydrated to form carbon monoxide (CO). A 27-year-old female was found unconscious inside a car, next to a container with a mixture of sulfuric acid and formic acid. Concentrations of up to 400 parts per million of CO were measured inside the car post ventilation. Serum carboxyhemoglobin level was 15% after receiving 100% oxygen for two hours. The patient received hyperbaric oxygen therapy after which she was extubated with normal mental status. On follow-up after three months, she demonstrated neurocognitive abnormalities suggestive of delayed neurological sequelae from CO exposure. [Clin Pract Cases Emerg Med. 2020;4(1):51–54.]

INTRODUCTION
Carbon monoxide (CO) toxicity is usually seen following exposure to smoke from house fires, heating system emissions, and exhaust fumes from motor vehicles. There are, however, various reported instances where CO produced through specific chemical reactions has been used by individuals to end their own lives. One such method involves combining formic acid with sulfuric acid, which produces CO. A total of 11 such reported cases were found in the literature, one of which was an outdoor occupational exposure. All the other cases were acts of suicide in enclosed surroundings including home spaces and, in one particular case, inside a car. We report a patient who generated CO from a sulfuric and formic acid mixture in her car in an attempt to commit suicide.

CASE REPORT
A 27-year-old female was found unconscious in the front seat of her car. On the car floor was a five-gallon (18.9 liter [L]) plastic drum containing a funnel and hose. The drum contained a green oily fluid assumed to be a mixture of sulfuric acid and formic acid because empty containers of the same were found in the vicinity of the scene. First responders measured CO levels of 400 parts per million (ppm) inside the car, which was measured after adequate ventilation. On-scene assessment documented that the patient was minimally responsive to pain with occasional tonic-clonic movements of her extremities. Initial vitals were a blood pressure of 192/125 millimeters mercury, heart rate of 135 beats per minute, respiratory rate of 24 breaths per minute and a Glasgow Coma Scale of 8/15 (best eye response 2, best verbal response 2, best motor response 4). She was started on 100% oxygen via a non-rebreather mask and transferred to the emergency department (ED).

In the ED, she was noted to be minimally responsive with tonic clonic movements of her distal extremities. This was followed by decerebrate rigidity. Her pupils were symmetric and reactive to light bilaterally. The remainder of her neurological exam was unremarkable. She remained tachycardic. The patient was eventually intubated for airway protection because of her depressed mental status. Her initial labs revealed an elevated carboxyhemoglobin (COHB) level of 15% (0-3%), lactate of 2 millimoles (mmol)/L (0.5-2.2 mmol/L) and a troponin level of 3.066 nanograms per milliliters (ng/ml) (0-0.03 ng/ml). The electrocardiogram was normal.

The patient was transferred to a hyperbaric center around four hours after being found on the scene and almost immediately underwent three sessions of hyperbaric oxygen therapy over 24 hours. The first session was at 2.8 ATA (atmospheres absolute) for 45 minutes, 2.0 ATA for...
60 minutes, and a five-minute air break. The second two
cycles were at 2.0 ATA for 90 minutes. She was extubated
the next day with normal mentation and neurological
exam. Around four weeks after discharge, her neurologic
evaluation demonstrated an anterograde amnesia beginning
with the suicide event. In addition, she demonstrated
other neurocognitive abnormalities suggestive of delayed
neurological sequelae (DNS). Brain magnetic resonance
imaging (MRI) showed abnormal restricted diffusion with
associated fluid-attenuated inversion recovery (FLAIR)
signal abnormalities in the white matter of the right temporal
lobe, bilateral globus pallidi, bilateral mesial temporal lobes,
hippocampus and scattered foci within the bilateral cerebellar
hemispheres suggestive of anoxic-ischemic brain injury.

DISCUSSION

Formic acid, when combined with sulfuric acid, gets
dehydrated to produce CO. This method is used in the
commercial production of CO in laboratories. In the past few
years, this method has been used to commit suicide. One
possible explanation could be the availability of a considerable
number of books, webpages, and online forums that provide
information on the production of CO for the purpose of
suicide.11-16 Interestingly, another chemical reaction that has
been recommended is heating calcium carbonate and zinc to
produce calcium oxide, zinc oxide, and CO.16 It is important
that first responders and emergency physicians be aware of
such chemical reactions so that they can deploy appropriate
on-scene and personal protection precautions.

Because CO is a colorless and odorless gas, it can
be hazardous to first responders on the scene. A previous
case report describes how a first responder developed CO
poisoning in similar circumstances requiring several days
of intensive critical care to recover.9 In most of the reported
cases, the victims had displayed warning signs outside
their enclosures, warning the first responders against CO
exposure. In our case, the first responders were initially
unaware of a potential hazardous materials (HAZMAT)
situation on the scene and hence were not adequately
protected with personal protective equipment. They reported
a “chemical smell” when they initially got into the car.
There were, however, no reported injuries among them on
later assessment. The HAZMAT team was later deployed
on the scene, and after ventilating the car they identified
the chemicals and measured the CO levels. Considering the
CO levels were measured after appropriate ventilation of
the car, the levels measured (400 ppm) were most likely an
underestimation of the maximum CO concentration.

The patient was not decontaminated on the scene
due to the initial lack of information about the HAZMAT
involved. The ED was alerted that the patient originated in
an unknown HAZMAT scene prior to patient arrival and
hence the patient was decontaminated with tepid water
and soap in a stand-alone decontamination room prior to
entering the ED.

The initial serum COHB level measured in the ED was
15.1%, more than two hours after the patient was started
on 100% oxygen. Considering that the half-life of COHB
is around 75 minutes on 100% oxygen at atmospheric
pressure,17 it is possible that the patient had a COHB
level in the range of 45% when she was found in the car,
assuming zero order kinetics.18 Use of a simplified version
of the Coburn-Forster-Kane model predicts that the patient
had a COHB level of 38.3 % [%COHB(%) = 100/[1 +
R(643/ppm CO)].19 Previous studies have interestingly
indicated poor correlation between initial COHB levels,
clinical manifestations, and the risk of delayed neurological sequelae. This patient would serve as an example to this fact, considering that the initial COHB levels were only moderately elevated despite the remarkable clinical presentation and noteworthy delayed neurological sequelae. It is also not clear how long the patient was exposed to CO inside the car before being evacuated, because the patient herself had poor recall of the events.

After resolution of the acute clinical course, the patient was admitted to psychiatry where she underwent treatment for depression. Four weeks after the exposure, she developed retrograde amnesia. She had no awareness of the suicide event or the events leading up to the suicide and demonstrated euthymia, inappropriate laughter, incongruent affect, childlike behavior, and impaired short- and long-term memory. She endorsed to having been treated for drug-resistant depression with ketamine infusions in the past but could not recall suicidality or plans of suicide around the time of the suicide attempt. She reported having no feelings of being suicidal a month after the event. At three months follow-up, many of her cognitive deficits had improved but her amnesia remained.

Delayed neurological sequelae following CO poisoning can present with a multitude of neurological and cognitive symptoms and signs. DNS may develop in up to 40% of the survivors of acute CO poisoning within 2-40 days. Cochrane systematic review analysis concluded that there is insufficient evidence to support the use of hyperbaric oxygen (HBO) to prevent DNS from CO poisoning. Similarly, the American College of Emergency Physicians has noted that there remains lack of clarity over whether HBO is superior to normobaric oxygen for improving long-term neurocognitive outcomes in CO exposure. Our patient developed DNS despite timely HBO therapy. This is the first case report of poisoning from chemical production of CO where long-term follow-up revealed delayed neurocognitive manifestations and distinct MRI findings in the face of expedient HBO therapy.

CONCLUSION

The case presented here demonstrates an unusual way of attempting suicide by combining formic acid with sulfuric acid to generate CO inside an enclosed car. The patient was treated with hyperbaric oxygen and survived the event but developed delayed neurological sequelae. Emergency physicians and first responders need to be aware of such chemical reactions to avoid injury to first responders and to guide appropriate treatment.

Documented patient informed consent and/or Institutional Review Board approval has been obtained and filed for publication of this case report.

Address for Correspondence: Muhammed Ershad, MD, Drexel University College of Medicine, Department of Emergency Medicine, Division of Medical Toxicology, 230 N. Broad St., Philadelphia, PA 19102. Email: me539@drexel.edu.

Conflicts of Interest: By the CPC-EM article submission agreement, all authors are required to disclose all affiliations, funding sources and financial or management relationships that could be perceived as potential sources of bias. The authors disclosed none.

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