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Pediatric Veno-Veno Extracorporeal Membrane Oxygenation Rescue From Carbon Monoxide Poisoning

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Background: Carbon monoxide poisoning affects approximately 5000 children per year and can be challenging to diagnose and treat (*Pediatr Emerg Med Pract*. 2016;13:1–24). It is in the differential diagnosis of a patient presented with altered consciousness. Patients may look quite "pink" and well perfused, but are often in serious distress. We present the first case in the literature of carbon monoxide poisoning treated with the use of veno-veno extracorporeal membrane oxygenation (ECMO).

Case: We report the case of a 10-year-old patient who had carbon monoxide poisoning (carboxyhemoglobin of 18%). She was treated with hydroxocobalamin at 70 mg/kg and was being prepared to transfer to a facility that offered hyperbaric therapy when she suffered a cardiac arrest requiring cardiopulmonary resuscitation. After 11 minutes of resuscitation, she had return of spontaneous circulation and an echocardiogram showed reasonable cardiac function. She was judged too unstable for ambulance transport and the ECMO team was called. Veno-veno ECMO was placed via a single right internal jugular dual-lumen catheter with fluoroscopy in the cardiac catheterization laboratory. There was a rapid improvement in carboxyhemoglobin level, and the ECMO therapy was weaned the next day. The patient eventually made a full recovery.

Conclusions: This is the first time that veno-veno ECMO has been reported for the emergent treatment of carbon monoxide intoxication. If emergency physicians are treating such a patient and cannot administer hyperbaric oxygen therapy, ECMO represents a valuable alternative that is not commonly thought of in this situation before.

Key Words: carbon monoxide, extracorporeal membrane oxygenation, poisoning, cardiac arrest

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Carbon monoxide poisoning affects approximately 5000 children per year in the United States and can be challenging to diagnose and treat.[1] The mainstay of treatment is oxygen therapy and supportive care, or hyperbaric oxygen therapy in severe cases. Severe intoxication can be fatal, however, even with these treatments. We present a case of carbon monoxide poisoning treated with the use of veno-veno (VV) extracorporeal membrane oxygenation (ECMO).

CASE

A 10-year-old, 40-kg African American girl was brought to the emergency department by first responders with carbon monoxide poisoning in her home due to a faulty furnace vent. She and a sibling were discovered by family and the patient was brought to our facility. The carbon monoxide meter reading was 500 ppm inside the home, and the period of exposure was unknown. Her younger brother was taken to another hospital but was pronounced dead on arrival. The time to get the patient from home to the emergency department was unclear. The patient was unresponsive upon arrival to the pediatric emergency department with a temporary airway in place; her blood pressure was 98/61 mm Hg; heart rate, 121 beats/min; respirations, 32 breaths/min, and a pulse oximeter saturation of 98%. The initial arterial blood gas (on 100% via ball-valve mask) showed a pH level of 7.06, partial pressure of carbon dioxide (pCO_2) of 56, partial pressure of oxygen (pO_2) of 517, bicarbonate of 13, base excess of −15, hemoglobin of 14.5, oxyhemoglobin of 82%, and carboxyhemoglobin of 18%. The child was endotracheally intubated and started on hydroxocobalamin at 70 mg/kg. She had cool clammy peripheral extremities. She was treated with volume infusion. The decision was made to transfer the patient to a facility to receive hyperbaric oxygen treatment, and the hydroxocobalamin was discontinued. The child, however, suffered from a cardiac arrest immediately before transfer. Pediatric advanced life support resuscitation was instituted for 11 minutes with return of spontaneous circulation. The patient was placed on an epinephrine infusion. The repeat arterial blood gas revealed a pH level of 7.03, pCO_2 of 63 mm Hg, pO_2 of 462 mm Hg, bicarbonate of 14 mEq/L, and base excess of −15 mEq/L. Because of the arrest, she was not deemed stable for transfer, and despite excellent pO_2, the ECMO team was called to the bedside. A rapid bedside echocardiogram was performed revealing normal heart function, and therefore, a swift multidisciplinary decision was made to place VV ECMO in the cardiac catheterization laboratory. A summary of her blood gas measurements is in Table 1.

PROCEDURAL DETAILS

The patient was brought emergently to the cardiac catheterization laboratory where she was prepped and draped in the usual sterile fashion and remained sedated under the care of a pediatric anesthesiologist. Venous access was obtained via the right internal jugular vein, and a 0.035 inch Amplatz Super-Stiff wire was advanced to the inferior vena cava guided by and confirmed with fluoroscopy. Progressive skin/soft tissue dilation was performed and a 23F catheter (7.7-mm diameter) bicaval cannula was advanced to the intrahepatic inferior vena cava, and the wire was removed after cannula position was confirmed with fluoroscopy. Wet-to-wet connections were made and VV ECMO circulation

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TABLE 1. Table of Arterial Blood Gas Measurements

Time	pH	pCO_2, mm Hg	pO_2, mm Hg	Carboxy Hgb, %	O_2 Sat, %
0833 h (May 3, 2016)	7.06	56	517	18	100
0905 h	7.03	63	462		100
VV ECMO placed in catheterization laboratory					
0932 h	7.20	28	424	6	100
~1015 h	7.36	19	430	2.7	
1428 h	7.57	20	536	2	100
0020 h (May 4, 2016)	7.43	37	325	2	100
0933 h	7.47	38	325	0	99

CarboxyHgb; carboxyhemoglobin; Sat, saturation.

was commenced with a Maquet Cardiohelp integrated oxygenator/rotary blood pump.

OUTCOME

After ECMO initiation, her blood gas revealed a pH level of 7.20, pCO_2 of 28 mm Hg, pO_2 of 424 mm Hg, and carboxyhemoglobin of 6%. The patient was transferred to the pediatric intensive care unit where the blood gas showed a pH level of 7.36, pCO_2 of 19 mm Hg, pO_2 of 430 mm Hg, base excess of −13 mEq/L, and carboxyhemoglobin of 2.7%. The patient's carboxyhemoglobin dropped to 0% after 21 hours of VV ECMO, and the ECMO was weaned off after a total of 24 hours of support.

Previous cases have used venoarterial ECMO to provide complete cardiopulmonary support, but this has complications particularly in small patients and may require an arterial cutdown. Veno-veno ECMO in this case was accomplished with a single jugular venous cannula to be used for therapy. The VV ECMO resulted in reduction of the carboxyhemoglobin and a rapid clinical stabilization. We cannot rule out spontaneous improvement without ECMO, but given her peri–cardiac arrest status, the likely cause of stabilization and improvement was ECMO.

The cannula was removed at bedside with placement of a single purse-string suture. An initial head magnetic resonance imaging (Fig. 1) showed typical carbon monoxide neurological injury. After a total of 21 days, the patient was discharged to a pediatric rehabilitation facility. She recovered complete physical and neurological function and is in regular classes for her grade and has not been admitted since.

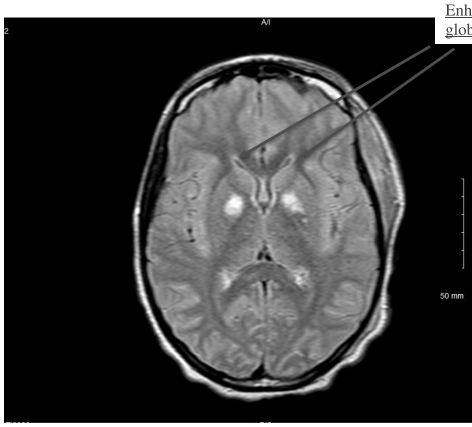

FIGURE 1. Enhancement of bilateral globus pallidus.

DISCUSSION

Carbon monoxide binds to hemoglobin, myoglobin, and intracellular cytochromes with a higher affinity than oxygen and hence significant exposure results in poisoning of the blood and cardiac muscle.[2,3] Lactic acidosis from poor tissue perfusion results, despite the often "rosy" pink complexion of the poisoning patient. Carboxyhemoglobin is a bright red color similar to oxyhemoglobin. Hyperbaric oxygen treatment is often considered in severe cases to prevent delayed neurological sequelae and results in displacement of carbon monoxide from hemoglobin by shifting the equilibrium to oxyhemoglobin instead.[4,5] Usually, mechanical ventilation with 100% oxygenation is not sufficient to displace the tightly bound carbon monoxide.

Extracorporeal membrane oxygenation has been used in rare cases, but all prior reports involve venoarterial ECMO.[6–9] Venoarterial ECMO involves a large central vein and artery and is associated with greater complexity and potentially higher risk of vascular complications, particularly in an emergent setting. Venoarterial ECMO is often needed because of cardiac collapse and severe ventricular dysfunction, which was not noted in this young patient.

Veno-veno ECMO can be performed with a single large (23F–31F catheter) venous cannula, as in the current report, or with 2 femoral venous cannulas. It is important to note that if the patient is unable to be moved to the cardiac catheterization laboratory, the placement of VV ECMO can be done at bedside particularly with imaging such as echocardiography. By using VV ECMO, we were able to rapidly remove carbon monoxide in a patient after arrest who was not a candidate for transfer to a facility with hyperbaric treatment. The ability to treat the patient without the necessity of a large arterial cannula and its attendant complications as well as removal of the venous dual-lumen cannula at bedside with a stitch represent significant advantages to this approach over those described previously.

CONCLUSIONS

The use of short-term VV ECMO has not been reported before and may represent an option in the care of these critically ill patients if other standard of care options is not available. It is applicable to pediatric and adult patients, but it requires the specialized team to be notified as soon as possible to allow for rapid evaluation and treatment.

REFERENCES

1. Macnow TE, Waltzman ML. Carbon monoxide poisoning in children: diagnosis and management in the emergency department. *Pediatr Emerg Med Pract*. 2016;13:1–24.
2. Rose JJ, Wang L, Xu Q, et al. Carbon monoxide poisoning: pathogenesis, management, and future directions of therapy. *Am J Respir Crit Care Med*. 2017;195:596–606.
3. American College of Emergency Physicians Clinical Policies Subcommittee (Writing Committee) on Carbon Monoxide Poisoning; Wolf SJ, Maloney GE, Shih RD, et al. Clinical policy: critical issues in the evaluation and management of adult patients presenting to the emergency department with acute carbon monoxide poisoning. *Ann Emerg Med*. 2017;69:98–107e6.
4. Huang CC, Ho CH, Chen YC, et al. Hyperbaric oxygen therapy is associated with lower short- and long-term mortality in patients with carbon monoxide poisoning. *Chest*. 2017;152:943–953.

5. Hui-Jun H, Dan-Feng F. Should hyperbaric oxygen be used in acute carbon monoxide poisoning? *Am J Emerg Med*. 2017;35:1030.
6. McCunn M, Reynolds HN, Cottingham CA, et al. Extracorporeal support in an adult with severe carbon monoxide poisoning and shock following smoke inhalation: a case report. *Perfusion*. 2000; 15:169–173.
7. Teerapuncharoen K, Sharma NS, Barker AB, et al. Successful treatment of severe carbon monoxide poisoning and refractory shock using extracorporeal membrane oxygenation. *Respir Care*. 2015;60: e155–e160.
8. Wang GS, Levitan R, Wiegand TJ, et al. Extracorporeal membrane oxygenation (ECMO) for Severe Toxicological Exposures: review of the Toxicology Investigators Consortium (ToxIC). *J Med Toxicol*. 2016;12:95–99.
9. Yin L, Cai Q, Zhen Q, et al. Treatment of acute carbon monoxide poisoning with extracorporeal membrane trioxygenation. *Int J Artif Organs*. 2012;35:1070–1076.