Neurologic recovery following cardiac arrest due to benzodiazepine and opiate toxicity.

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Letter to the Editor

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Sir,

We present a unique case of a patient who received therapeutic hypothermia (TH) following cardiac arrest due to opiate and benzodiazepine toxicity and achieved good neurologic recovery.

A 29-year-old man with a medical history of substance abuse and bicuspid aortic valve was observed using heroin and benzodiazepines 5 h prior to suffering a pulseless electrical activity (PEA) arrest. He was last seen in usual state of health 30 min prior to Emergency Medical Services (EMS) arrival. Prehospital care included: cardiopulmonary resuscitation, endotracheal intubation, and the administration of naloxone, epinephrine, atropine, bicarbonate and amiodarone. He experienced return of spontaneous circulation (ROSC) with a sinus mechanism at 84 beats per minute and a systolic blood pressure of 80 mmHg. Given his coma, paramedics initiated cold saline while enroute to the hospital. In the emergency department (ED), his Glasgow Coma Score (GCS) was four (Eye-one, Verbal-one, and Motor-two) and core temperature was 32 °C. Corneal, pupillary, and cough reflexes were preserved. Brain computed tomography demonstrated bilateral globus pallidum hypodensities (present on follow-up brain magnetic resonance imaging—Fig. 1). ED laboratory studies revealed: pH 6.86 (7.32–7.43), pCO_2 81 mmHg (41–51 mmHg), HCO_3 14 mEq/L (22–26 mEq/L), lactate 14.5 mMol/L (0.5–1.6 mMol/L), AST 1551 IU/L (<40 IU/L), ALT 2555 IU/L (<40 IU/L), BUN 21 mg/dL (5–20 mg/dL), creatinine 3.5 mg/dL (0.5–1.4 mg/dL), troponin I 0.88 ng/ml (<0.1 ng/ml), and CPK 4592 IU/L (<200 IU/L). Toxicology testing demonstrated benzodiazepines and opiates. Protocolized post-cardiac arrest care including intravenous cold saline and external cooling maintained the patient's core temperature between 32 and 34 °C [1]. After 24 h, he was re-warmed at 0.25–0.5 °C/h. On hospital day (HD) three, his GCS was five (Eye-one, Verbal-one, and Motor-three), and on HD five his GCS was six (Eye-one, Verbal-one, and Motor-four). The patient awoke and followed commands on HD six. He was extubated on HD ten. His hospital course was complicated by rhabdomyolysis with peak CPK of 26,804 IU/L, oliguric renal failure necessitating dialysis, mild dysarthria and cognitive deficit. After medical stabilization, he was transferred to an inpatient rehabilitation facility on HD 13. At eight months follow-up, he attends drug rehabilitation, lives independently, works, and runs three to four days per week. His Cerebral Performance Category (CPC) is one.

Patients suffering from toxin-mediated cardiac arrest follow a similar pathway of neurologic injury as those with primary ventricular tachycardia/fibrillation (VT/VF). Animal studies demonstrating the beneficial effects of TH on neurological recovery have been conducted primarily after asphyxiation-induced PEA or asystolic cardiac arrest [2,3]. While the strongest benefit of TH likely relates to cerebral metabolic and edema changes, additional benefits to cooling intoxicated patients may exist. In animals, hypothermia increases the LD50 of ethanol, heavy metals, methylmercury, and pesticides despite decreasing drug clearance [4].

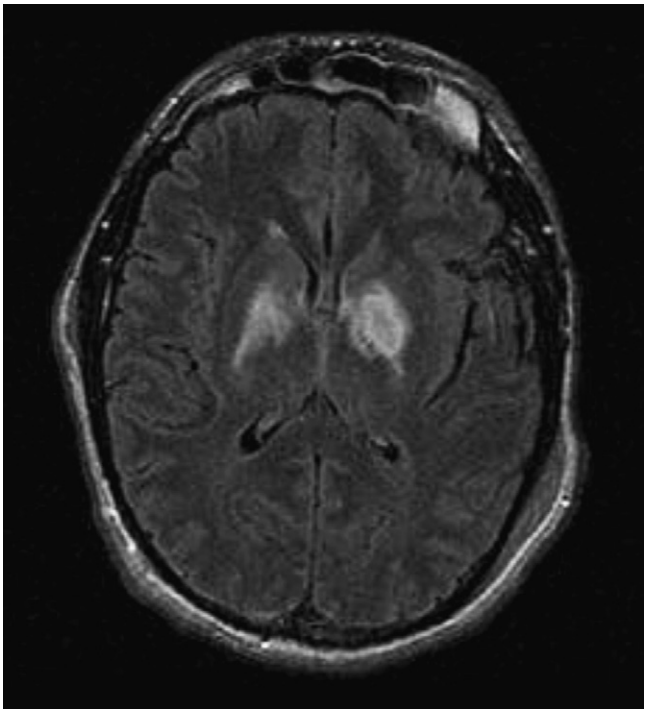

Fig. 1. Magnetic resonance imaging of patient demonstrating bilateral basal ganglia infarction.

TH improves neurologic outcome following cardiac arrest [1]. The American Heart Association and International Liaison Committee on Resuscitation recommend this therapy in unresponsive patients with ROSC following out-of-hospital VT/VF arrests [5]. We suggest that TH following cardiac arrest may benefit comatose victims of toxin-mediated cardiac arrest.

Conflict of interest statement

The authors have no conflict of interest.

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