Poppers-induced Life-Threatening Methemoglobinemia

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Figure 1. (A) Extensive gray discoloration of the skin after ingestion of recreational poppers. (B) Chocolate-brown color of a venous sample suggestive of severe methemoglobinemia (here, 75.4%). (C) Normal skin recoloring was obtained rapidly after intravenous infusion of methylene blue.

A 25-year-old woman was brought to the emergency department in a coma (Glasgow coma score: 5) after ingesting ethanol and poppers at a party. On admission, she was in respiratory distress (respiratory rate = 30/min; oxygen saturation as measured by pulse oximetry = 89% on 15 L/min oxygen by face mask), was tachycardic (130/min) with preserved blood pressure, and had extensive gray skin discoloration (Figure 1A). Cardiopulmonary auscultation and abdomen examination were unremarkable. Arterial blood gas analysis showed pH = 7.29, PaO₂ = 365 mm Hg (48.7 kPa), PaCO₂ = 34 mm Hg (4.5 kPa), bicarbonate = 16 mmol/L, and lactate = 7.0 mmol/L ($n < 2.0$). Extremely high methemoglobin levels (75.4%) were measured. Venous blood collected for laboratory tests showed a chocolate-brown color characteristic of severe methemoglobinemia (Figure 1B). She promptly received high-flow nasal oxygen (FiO₂ = ~100%) and 2 mg/kg methylene blue infusion, resulting in her rapid awakening and resolution of her tachypnea, skin discoloration, acidosis, and methemoglobinemia within 2 hours (Figure 1C). The blood ethanol concentration was 1.46 g/L. She was discharged 8 hours after admission.

Poppers are widely available bottled volatile nitrites that are inhaled for recreational purposes in relation to their vasodilatory, muscle-relaxant, and euphoria-inducing properties (1). Direct hemoglobin-oxidizing effects may induce life-threatening methemoglobinemia, at higher risk by ingestion, as in the case with the highest recorded value of 94% (2). Management requires appropriate diagnosis and timely antidote administration. Antidotes with demonstrated efficacy in reversing methemoglobinemia include methylene blue and ascorbic acid (3, 4). However, the therapeutic effects of ascorbic acid are delayed (up to 24 h). Hence, its use should be reserved for cases where methylene blue is contraindicated, e.g., known glucose-6-phosphate dehydrogenase deficiency, or when methylene blue is unavailable. ■

Author disclosures are available with the text of this article at www.atsjournals.org.

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