Carbon Monoxide Toxicity in the Critically Ill patient

Key Article

Chiew AL, Buckley NA. Carbon monoxide poisoning in the 21st century. Critical Care. 2014, 18:221.

Introduction

- One of the most common unintentional toxic exposures in the developed world
- In the US, home CO alarms, education have significantly reduced the annual death rate from 1,000-2,000/year
- However, there are a large number of annual exposures – 50,000/year due to home heating systems, generators in addition to intentional exposures from car exhaust, charcoal burning heat devices, and catalytic converters.
- Acute toxicity can lead to a number of clinically significant short and long-term sequelae that must be managed and considered in the ED.

Mechanisms of toxicity

- Tissue hypoxemia due to reduced O2 carrying capacity – CO binds to hemoglobin with an affinity of 200-240 times that of O2.
- COHb prevents O2 binding he hemoglobin, but also increases hemoglobin's affinity to hold on to O2 at remaining carrying sites.
- CO causes cellular injury:
    - Cellular mitochondrial dysfunction
    - Endothelial oxidative stress, lipid peroxidation, and inflammation: Likely responsible for delayed neurologic sequelae
    - Impaired ATP synthesis
    - Reperfusion injury with treatment
- Common organs most severely affected – those with the highest oxygen demand
    - CNS
    - Cardiac

Toxicity assessment

- Severity of poisoning is a function of CO *dose:* Duration of exposure + air concentration of CO.
- COHb concentrations are only a rough guide to severity of exposure
    - < 20% COHb: minimal symptoms
    - 20-40% COHb: Headache, tachycardia, confusion, nausea/vomiting, syncope
    - 40-60% COHb: Coma, seizure, ECG changes, arrhythmias/ST changes
    - > 60% COHb: Cardiopulmonary failure, death
- **Beware**: Similar to Tylenol levels, a first measured COHb is not a reliable way to measure toxicity severity or predict long-term outcome
    - Multiple patient modifiers can affect the patient specific toxicity level and elimination half-life.
        - Anemia or other hemoglobinopathies such as sickle cell, thalassemia, etc.

        - Significant coronary or cerebrovascular disease
        - These patients with may experience significant toxicity at lower COHb levels.
- Devices being developed to detect pulse CO oximetry have failed to pass a number of FDA regulations and cannot be recommended for triaging purposes or clinical use.
- **CHECK A POC VBG at a minimum**
    - Venous COHb levels correlate within 1-2% of arterial samples
    - Can alternatively use co-oximetry if a POC device not available
- Can consider other laboratory testing to assess for other organ ischemia or risk modifiers
    - CBC
    - BMP
    - LFTs
    - CK
    - Other tox labs (ASA, APAP, ethanol, etc.); CN level in housefires

- Neurologic and cardiac signs & symptom assessment remain the standard in determining the severity of exposure.
    - HPI
        - Severe cases: coma, seizures, severe metabolic acidosis, heart failure
        - Awake patient: Syncope & Chest pain are most concerning
        - **Prolonged LOC, age > 36 years old, and COHb level > 25% appear to be the best predictors of those who may go on to develop delayed neurologic sequelae and may benefit from HBO therapy.**
    - Early ECG, troponin
    - Focused US may find significant CV effects
        - Reduced cardiac function
        - Pulmonary edema
        - One prospective study of TTE on 40 pts with CO exposure found that 50% had significant LV systolic dysfunction, with whom a majority recovered within 72 hours.

Management

- Aside from removing the patient from the source, should also include
    - Bread and butter EM Care – A-B-C's
    - 100% O2 by NRB for at least 4-6 hours in severe cases
    - **A couple important NOTES**:
        - If intubating the patient, continue 100% FiO2 as well!
        - Serial ECGs and cardiac enzymes in patients with a history of LOC, cardiovascular disease, chest pain, or ECG changes.
    - Remember co-existing cyanide toxicity in housefire patients
- Half-life of CO is:
    - About 4-5 hours for patients breathing Room Air at 1 ATM (sea-level)
    - About 40-80 minutes with 100% FiO2 NRB
    - About 23 minutes with HBO at 2 ATM* - this is an important clinical consideration as far as how HBO may actually benefit CO patients

- Hyperbaric oxygen therapy (HBO)
    - Most beneficial effect may be at the prevention of long-term neuropsychiatric sequelae

- HBO therapy does not appear to impact mortality
- There really aren't any standard HBO therapy algorithms
  - Should occur within 12-hours of exposure
  - Most are patients receive between 45 min- 3 hours per dive
  - Patient may receive a single or multiple sessions
- Who to refer to an HBO center?
  - ACEP: HBO cannot be mandated, no clinical variables (including COHb levels) can help find subgroup of benefit
  - NIH: do not recommend treatment, insufficient evidence
  - Undersea and Hyperbaric medical Society:
    - Patients with syncope, abnormal neurologic signs, cardiovascular dysfunction, severe acidosis
    - Age > 36 years old with first COHb level of > 25%
    - Pregnant patients: fetus is at particular risk due to higher affinity of fetal hemoglobin for CO.

Take home points

- Severity of CO toxicity should be assessed based on symptoms and presentation, not just by level.
- 100% FiO2 via Non-rebreather mask remains standard of care, but remember to keep patient on 100% FiO2 even after intubation
- Most POC VBGs will provide enough information to risk stratify a patient with CO toxicity
- HBO therapy is certainly not standard of care, however, patients at high risk including those with a loss of consciousness, CV dysfunction, older, or pregnant should probably be transferred.