IAEM Clinical Guideline

Acute Carbon Monoxide Poisoning

Version 1.0

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In collaboration with the IAEM Clinical Guideline Development Committee

To reference this document please reference to:


DISCLAIMER

IAEM recognises that patients, their situations, Emergency Departments and staff all vary. These guidelines cannot cover all clinical scenarios. The ultimate responsibility for the interpretation and application of these guidelines, the use of current information and a patient's overall care and wellbeing resides with the treating clinician.
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GLOSSARY OF TERMS

CK  Creatinine Kinase
CO  Carbon Monoxide
COHb  Carboxy-haemoglobin
ECG  Electrocardiogram
ED  Emergency Department
FiO2  Fraction of Inspired Oxygen
GCS  Glasgow Coma Scale
LOC  Loss of Consciousness
O2  Oxygen
Acute Carbon Monoxide Poisoning

INTRODUCTION

Carbon Monoxide poisoning is the most common human poisoning. CO is formed by incomplete combustion. Smoke inhalation from fires, motor vehicle exhausts and other engine exhausts are the most prevalent sources of exposure.

Approximately 1% to 3% of all poisonings are fatal.¹ Those at highest risk of mortality are:

- Older patients (with fire as source of CO)
- Patients with very elevated COHb levels and respiratory failure
- Patients who experience loss of consciousness

Diagnosis is based on the clinical triad of:

- History of CO exposure,
- Elevated COHb levels, AND
- Symptoms consistent with CO poisoning.
PARAMETERS

Target audience Medical professionals working in Emergency Departments.

Patient population Patients >18 years of age presenting with acute CO poisoning, diagnosed or suspected.

Exclusion criteria Pregnant patients and foetal exposures.
Chronic CO poisoning.

AIMS

The aim of this document is to provide guidance to clinical staff involved in the first line assessment and management of adults presenting to the ED with suspected or diagnosed acute CO poisoning.
CLINICAL FEATURES

Clinical features vary from mild and non-specific through to life threatening (Table 1).

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<th>Symptoms and signs</th>
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<tr>
<td>General</td>
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<tr>
<td>Headache (90%), nausea, vomiting, weakness</td>
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<tr>
<td>Neurological</td>
</tr>
<tr>
<td>Dizziness, confusion, ataxia, seizures, coma</td>
</tr>
<tr>
<td>Cardiovascular</td>
</tr>
<tr>
<td>Sinus tachycardia, arrhythmias, myocardial infarction, pulmonary oedema</td>
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</tbody>
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*Table 1: Clinical Features of CO poisoning*

INVESTIGATIONS

Patients with suspected exposure to CO should have their COHb level measured. The gold standard test is laboratory blood gas CO-oximetry².

This can be drawn from venous blood.

- Levels >3% are abnormal in non-smokers
- Levels >10% are abnormal in smokers
- Severe poisoning occurs at 30%

Other investigations can be considered to identify complications of CO poisoning (Table 2)

<table>
<thead>
<tr>
<th>INVESTIGATIONS</th>
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<tr>
<td>ECG / Troponin</td>
<td>Myocardial infarction</td>
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<td>Renal function tests, Liver function tests</td>
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<td>CT Brain / MRI Brain</td>
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<td>CK</td>
<td>Rhabdomyolysis</td>
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<tr>
<td>Blood Glucose level</td>
<td>Hyperglycaemia</td>
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</table>

*Table 2: Other investigations considered in CO poisoning*
MANAGEMENT

- Supportive care: patients with altered GCS or cardiovascular instability should be managed in a resuscitation area with full monitoring.

- Provision of high concentration $O_2$ as quickly as possibly is the mainstay of treatment. For non-intubated patients, $O_2$ should be provided at 15L/minute via a tight fitting non-rebreather mask with reservoir bag. Consider high flow nasal oxygenation to administer 100% $O_2$ at 50-60L/min if available. Intubated patients should have $FiO_2$ of 1.0. The endpoint of $O_2$ therapy remains poorly defined. Duration of $O_2$ therapy should be at least 6 hours. COHb level should be <5% and patient should be asymptomatic prior to cessation of $O_2$ therapy in the ED.

- Hyperbaric $O_2$ therapy: Evidence for the use of hyperbaric $O_2$ therapy in CO poisoning remains uncertain. It should be considered, in consultation with a clinical toxicologist, in patients with the following:
  - Loss of consciousness
  - Ischaemic cardiac changes
  - Neurologic deficits
  - Significant metabolic acidosis
  - COHb levels >25%

The National Hyperbaric Medicine Unit is located at University Hospital Galway. For emergencies, contact the University Hospital Galway switchboard at 091-524222 and ask for the third on-call anaesthetist.

- Consider cyanide poisoning in any patient who has been exposed to smoke inhalation.

- Hyperglycaemia, if present, should be treated with insulin, as neurological outcomes following CO poisoning may be worse in patients with hyperglycaemia.
SPECIAL CONSIDERATIONS

Survivors of CO poisoning have a higher long-term mortality rate than their non-poisoned counterparts, and 15% to 40% suffer from near-permanent neurocognitive sequelae. These patients require specialist follow-up.

OTHER RESOURCES

- Toxbase - The primary clinical toxicology database of the National Poisons Information Service, available at https://www.toxbase.org
- National Poisons Information Centre at Beaumont Hospital, Dublin 9 at 01-809 2566 or 01-837 9964 (24 hours)
REFERENCES


4. https://bestpractice.bmj.com/topics/en-us/432