Carbon Monoxide Poisoning

Category

Symptoms

Chronic carbon monoxide poisoning is frequently undiagnosed since the features are non specific. It is often associated with the use of faulty gas heaters in unventilated areas and therefore occurs more commonly in the winter months. Features include headache, nausea and flu-like symptoms. The diagnosis should be considered particularly if there are other members of the same house experiencing similar symptoms.

Acute carbon monoxide poisoning should be considered in victims from house fires or with self poisoning from car exhausts.

Headache, nausea, irritability, weakness and tachypnoea followed by dizziness, ataxia, agitation, impairment of consciousness and respiratory failure. Cerebral oedema and metabolic acidosis may develop in serious cases. Cherry red skin appearance is a very late and uncommon seen sign.

Less common features include: skin blisters, rhabdomyolysis, acute renal failure, pulmonary oedema, myocardial infarction, retinal haemorrhages, cortical blindness, choreoathetosis, and mutism.

Indicators of severity

Any new objective acute neurological signs e.g. increased tone, upgoing plantars
Coma
Need for ventilation
ECG showing infarction or ischaemia
Clinically significant acidosis
Initial carboxyhaemoglobin > 30%

But note that the link between carboxyhaemoglobin level and outcome is weak

Management

- Remove from exposure.
- Maintain a clear airway and adequate ventilation. Have a low threshold for intubation and ventilation.
- Give oxygen in as high concentrations as possible using a tightly fitting face mask and a non re-
breathing bag.

- Monitor the heart rhythm.
- Investigations: FBC, U&E, Carboxyhaemoglobin level, cardiac enzymes, ABGs, ECG, CXR. A carboxyhaemoglobin level of 20% indicates significant exposure. However, concentrations less than this do not exclude significant poisoning and the relationship between the carboxyhaemoglobin level and the severity of poisoning and/or clinical outcome is poor.
- Metabolic acidosis should be corrected by increasing oxygen delivery to the tissues. The use of intravenous sodium bicarbonate may make this more difficult and is therefore best avoided.
- If lactate >10, consider cyanide poisoning.

**N.B. Pulse oximeter will give a falsely elevated reading for SpO₂.**

**Consider Hyperbaric oxygen treatment for the following:**

1. Loss of consciousness at any time.
2. In patients who have been unconscious look for extrapyramidal features and retinal haemorrhages to assess the severity of CNS toxicity.
3. The role of hyperbaric oxygen therapy has been controversial. However, it may be considered in patients with loss of consciousness, CNS features of poisoning, cardiac ischaemia or metabolic acidosis (Weaver, 2002).
4. Discuss with ITU/Anaesthetic Registrar.

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