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CASE REPORT

Acute Dyspnoea with Co-Incidental Finding of Carbon Monoxide Poisoning - A Case Report with Review of Current Literature

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Abstract

Introduction: Carbon monoxide poisoning can present in subtle ways with a variable range of symptoms from mild headaches to profound neurological impairment. The primary aim of this article is to highlight the importance of early detection of carbon monoxide poisoning through meticulous clinical assessment. The secondary aim of this article is to review the current treatment strategies.

Case Presentation: A 73-year-old gentleman was brought in by ambulance to the emergency department with acute dyspnoea on a background of chronic obstructive pulmonary disease (COPD). In addition, he had symptoms of headache and general malaise and was noted to have carbon monoxide poisoning. He was noted to have both carbon monoxide poisoning and worsening carbon dioxide retention. He was successfully treated with Non-invasive ventilation (NIV) with a good outcome and return to pre-morbid state.

Discussion: The emergency physician is at the forefront to assess clinically and detect the presence of carbon monoxide and institute the correct management. The superiority of the use of hyperbaric oxygen therapy (HBOT) for acute carbon monoxide poisoning over normobaric oxygen remains controversial.

Conclusion: The early detection and appropriate management of carbon monoxide poisoning is important as symptoms may be subtle. Further studies are required to compare the superiority of HBOT over normobaric oxygen therapy and extended follow up to assess long term neurological sequelae.

Keywords

Carbon monoxide poisoning, Non-invasive ventilation, COPD, Clinical assessment

Introduction

Carbon monoxide poisoning can present in a subtle form which may not be apparent to the emergency physician unless specifically investigated. Carbon monoxide poisoning is still a leading cause of death by poisoning in both the United States and United Kingdom causing upto 50,000 deaths per annum [1]. There are various stages of carbon monoxide poisoning and the symptomatology changes accordingly. These can range from headache to loss of consciousness. There are various sources where there is incomplete of fuel including faulty heaters which are the primary source. Carbon monoxide being odourless makes it difficult to detect. In cases of sublethal carbon monoxide exposure, the predominant signs are likely to neurological with sharpened cerebellar signs [2].

Purpose of this Article

The primary aim of this article is to highlight the importance of early detection of carbon monoxide poisoning through meticulous clinical assessment. The secondary aim of this article is to review the current treatment strategies.

Case Presentation

A 73-year-old gentleman was brought in by ambulance to the emergency department with acute dyspnoea on a background of chronic obstructive pulmonary disease (COPD). In addition to the dyspnoea, he complained of a generalised mild headache and general malaise both of which were new for the previous 2 days. He was

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noted to be in decompensated type 2 respiratory failure and evidence of carbon monoxide poisoning.

Past medical history

Our patient had a few co-morbidities including-Longstanding diagnosis of COPD with type 2 respiratory failure, secondary polycythaemia, previous pericarditis, oesophagitis, duodenal ulcer and cervical spondylosis.

During assessment, it transpired that our patient had a limited exercise tolerance capacity due to his longstanding airway disease. This led to discussion with him and our intensive care colleague about ceiling of care. It was noted that he had a 'Do not attempt cardiopulmonary resuscitation' (DNACPR) in his case notes. During the initial consultation, our patient stated on many occasions that he did not want CPR.

His medication included beta agonist inhalers; carbocisteine and oral proton pump inhibitor tablets. There were no known drug allergies.

Examination findings and investigations

On examination, the gentleman was in mild respiratory distress with minimal wheeze bilateral on auscultation without any evidence of focal consolidation.

Whilst examining our gentleman we noticed that his trousers were partially burnt when asked he recently had a heater fitted. With the symptom of headache, concern of carbon monoxide poisoning was raised which was confirmed on arterial blood gas.

Aside from these findings, there were no additional findings on examination.

Blood tests- all noted to be largely unremarkable. See (Table 1) for arterial blood gas results.

Radiograph of the chest revealed changes related to longstanding chronic obstructive airway disease and bronchial wall thickening. There was no evidence of focal changes.

Discussion

In our case our patient had evidence of both decompensated Type 2 respiratory failure and evidence of mild carbon monoxide poisoning. He was noted to have carbon dioxide retention with evidence of mild respiratory acidosis on his initial arterial blood gas. There was evidence of long term renal compensation indicated by a raised bicarbonate level. Having had a discussion with him, he was subsequently started on continuous bilevel positive airway pressure ventilation (BPAP) in an attempt to improve his arterial carbon dioxide level and improve his acid base status. On his previous admissions he had responded to non-invasive ventilation. It transpired that the cause of carbon monoxide was due to a faulty gas heater that had recently been fitted. In order to ensure the safety of his wife, she was contacted and appropriate safety measures were taken.

On medical high care, he had BIPAP overnight which improved his blood gases and his breathing improved back to his baseline, following which he was safely discharged home with advice.

Patients with underlying cardiovascular disease, the elderly, the pregnant patient are particularly predisposed with a higher incidence of adverse outcome. The mainstay of treatment is hyperbaric oxygen, however the widespread availability to such facilities is limited.

Carbon monoxide has a greater affinity (upto 200 times [3]) to bind to the haem groups in the haemoglobin complex compared to oxygen. This leads to formation of carboxyhaemoglobin (CoHb). It is also implicated in an inflammatory response which can have a delayed impact on the neurones leading to long-term neurological sequelae.

Hyperbaric oxygen therapy (HBOT)

The purpose of hyperbaric oxygen therapy (HBOT) is to competitively bind to the haem group and displace the carbon monoxide molecules. To our knowledge there are no definite cut-off levels of carbon monoxide level at which HBOT should be instituted.

With regards to treatment, if our patient did not respond to the therapy that we initially instituted then he perhaps would require hyperbaric oxygen. Alternatively, if there was any evidence of significant neurological impairment that would be an indication for hyperbaric oxygen. Indications for HBOT would include in those with loss of consciousness or with evidence of significant poisoning.

In a 2011 Buckley, et al. [4], analysed the use of hyperbaric oxygen for carbon monoxide poisoning through systematic review of studies. The authors came to the conclusion that there was insufficient evidence to support the role of hyperbaric oxygen in limiting the incidence of adverse neurological outcomes. The outcome from the study was to have wider study groups across multi-centres.

Table 1: Arterial blood gas results.

Values	Baseline (last admission)	Prior to NIV	On BIPAP (1 hour)
FiO_2 and flow/min	24% on 2 l	28% on 4 l	48%, 20/4 (IPAP/EPAP)
H^+	45.5	47.9	47.3
$PaCO_2$	8.50	9.00	8.12
PaO_2	8.55	7.35	8.62
HCO_3^-	32.9	34	34
CoHb (0.5-2.5%)	0.5	**10.0**	**8.0**

High flow oxygen therapy may have had implications as hypoxic drive would have significantly diminished in our patient who had chronic carbon dioxide retention. A further discussion of the effects of hyperbaric oxygen therapy in COPD with chronic carbon dioxide retention is beyond the remit of this article. Additionally, a further discussion of NIV in the management of carbon monoxide is not included in this article.

NICE (National Institute for Health and Care Excellence)

The guidance was updated in 2013 [5] and alerts the clinician to signs of possible carbon monoxide poisoning. With regards to treatment in significant CoHb poisoning, high flow oxygen therapy is advised with correction of hypoglycaemia. The guidance is also to contact the UK National Poisons Information Service (NPIS) for specialist advice.

Review of current literature

The ACEP (American College of Emergency Physicians) produced an initial clinical policy in 2008 [6] which looked into the role of HBOT in CO poisoning. It suggested that even though HBOT is a recognised therapeutic option, its use could not be justified in all cases.

The committee produced a further clinical policy in 2017 [7] answering critical questions comparing normobaric oxygen therapy with HBOT. In the article the authors reviewed all the studies comparing the two treatment options, the recommendation was to use HBOT in the acute poisoning however the superiority of HBOT over normobaric oxygen therapy is unclear. In addition to the above recommendation, further research is needed over longer periods of time to truly assess the long term neurological implications.

Role of erythropoietin (EPO)

Studies have shown that the early use of EPO [8] reduces the neurological outcome and potentially reduce the long term adverse neurological sequelae. There are numerous animal studies proposing the use of EPO in CO poisoning and the potential neuroprotective effects. Further studies will be needed in humans to supports its routine use in significant carbon monoxide poisoning.

Although our patient received NIV in this case, the basis of discussion of this case is not necessarily to compare NIV with HBOT. This could potential be research topic comparing the efficacy of NIV with HBOT in the treatment of CO poisoning.

Conclusion

Through this article we have highlighted various issues namely: -

- The ceiling of care in the acutely dyspnoeic patient including any prior resuscitation plans.
- The early identification of carbon monoxide poisoning through meticulous history taking and examination.
- The awareness of the non-specific nature of presentation of carbon monoxide poisoning and the established treatment modalities.

The routine use of arterial blood gases to detect high carbon monoxide level should be instigated as it remains the gold standard means of detection. It is also worth noting that blood carbon monoxide levels may be marginally raised in smokers and in houses where gas appliances are used. It is the correlation between the raised carbon monoxide level and clinical findings that need to be taken into account.

Whilst reviewing the available literature, we have noted that although there is a role of HBOT in acute carbon monoxide poisoning there is no definite superiority of HBOT over normobaric oxygen therapy. Further well-designed studies with the appropriate length of follow up is required.

Ethics

There was no requirement for ethical approval of this article, our patient had given consent.

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Conflicts of Interest

The authors of this article have identified no conflicts of interest.

References

1. HMSO (1996) Mortality statistics cause: England and Wales (Series DH2 No23). Office for National Statistics, London.
2. Notermans NC, Van Dijk GW, Y van der Graaf, J van Gijn, JH Wokke (1994) Measuring ataxia: Quantification based on the standard neurological examination. J Neurol Neurosurg Psychiatry 57: 22-26.
3. Piantadosi CA (1999) Diagnosis and treatment of carbon monoxide poisoning. Respir Care Clin N Am 5: 183-202.
4. Nick A Buckley, David N Juurlink, Geoff Isbister, Michael H Bennett, Eric J Lavonas (2011) Hyperbaric oxygen for carbon monoxide poisoning. Cochrane Database Syst Rev 13: CD002041.
5. https://cks.nice.org.uk/carbon-monoxide-poisoning#!topic-summary
6. Wolf SJ, Lavonas EJ, Sloan EP, Jagoda AS, American College of Emergency Physicians (2008) Clinical policy: Critical issues in the management of adult patients presenting to the emergency department with acute carbon monoxide poisoning. Ann Emerg Med 51: 138-152.
7. Stephen J Wolf, Gerald E Maloney, Richard D Shih, Bradley

D Shy, Michael D Brown (2017) Clinical Policy: Critical issues in the evaluation and management of adult patients presenting to the emergency department with acute carbon monoxide poisoning. Annals of Emergency Medicine 69: 98-107.

8. Pang L, Bian M, Zang XX, Wu Y, Xu DH, et al. (2013) Neuroprotective effects oferythropoietin in patients with carbon monoxide poisoning. J Biochem Mol Toxicol 27: 266-271.