

A case of extreme carboxyhaemoglobinemia due to vaping

Audrey K. Grech¹ | Dominic T. Keating^{1,2} | Daniel J. Garner¹ | Matthew T. Naughton^{1,2} 

¹Department of Respiratory Medicine, Alfred Health, Melbourne, Victoria, Australia

²Department of Medicine, Monash University, Melbourne, Victoria, Australia

Correspondence

Matthew T. Naughton, Department of Respiratory Medicine, Alfred Hospital, 55 Commercial Road, Melbourne, VIC 3004, Australia.
Email: m.naughton@alfred.org.au

Associate Editor: Diego Castillo Villegas

Abstract

Acute carbon monoxide (CO) poisoning is known to cause neurological, metabolic and cardiorespiratory sequelae. However, data on chronic CO exposure are scant, particularly in the context of vaping, which recent literature suggests may be a greater source of CO than tobacco cigarette smoking. During a series of admissions at the time of vaping, our patient repeatedly presented with significant CO poisoning and developed pulmonary arterial hypertension with resultant high-output right heart failure. On each occasion, our patient's levels of carboxyhaemoglobin were both higher and took longer to resolve than 12 smokers who underwent arterial blood gas testing at two time points. Our observation may reveal an association between vaping, chronic carboxyhaemoglobinemia and the development of cardiorespiratory disease. Thus, further studies into the safety of vaping and chronic CO exposure are urged.

KEYWORDS

carboxyhaemoglobin, e-cigarette, heart failure, pulmonary hypertension, vaping

INTRODUCTION

We present a case of extreme carboxyhaemoglobinemia in a middle-aged female with a significant smoking and vaping history.

CASE REPORT

A 52-year-old woman presented to our hospital on 12 occasions over 8 years, 11 admissions were in the last 2 years of her life. All presentations were similar with symptoms of dyspnoea, lower respiratory tract infection, weakness, atypical chest pain, headache and drowsiness, with only her last admission requiring intensive care. She volunteered a significant vaping history, using one bottle of light and mildly sweet (nicotine 6 mg/ml) e-liquid (Black Note Inc, Irvine, California) per fortnight for over 1 year, as well as up to 3 packets of cigarettes per day since the age of 16, with past marijuana use. The type of vaping device used is unknown. Furthermore, it should be noted that her partner vaped in her presence using the same device prior to her taking up vaping. Her comorbidities included: morbid obesity (body mass index of 37 kg/m²), severe obstructive sleep apnoea,

chronic obstructive pulmonary disease (COPD), ischaemic heart disease, polycythaemia secondary to chronic hypoxia, chronic schizophrenia and epilepsy. Her Charlson Comorbidity Index score was 4, with a predicted 10-year survival of 53%. She was treated with carbamazepine and flupentixol, had regular venesection and used continuous positive airway pressure irregularly due to poor tolerance. On each admission, examination revealed Glasgow Coma Scale of 14, fluid overload, tachypnoea, normal blood pressure, sinus tachycardia and hypoxia, with SpO₂ readings as low as 66% on room air.

Her investigations revealed hypercapnia (partial pressure of carbon dioxide [PaCO₂] range: 51–60 mmHg) without lactic acidosis, elevated brain natriuretic peptide (485–962 ng/L) and pulmonary arterial hypertension (mean pulmonary artery pressures of 26–45 mmHg) with high cardiac output (7.4–9.8 L/min). Exacerbations were commonly characterized by minor areas of pulmonary consolidation without pulmonary emboli on imaging. Lung function tests once clinically stabilized indicated a mixed picture of severe airflow obstruction and restriction with a reduced diffusion capacity and gas trapping. Her carboxyhaemoglobin (COHb) was grossly elevated on arterial blood gas (ABG), ranging from 7.1% to 32.9% (Figure 1). Her COHb levels

This is an open access article under the terms of the Creative Commons Attribution-NonCommercial-NoDerivs License, which permits use and distribution in any medium, provided the original work is properly cited, the use is non-commercial and no modifications or adaptations are made.

© 2022 The Authors. *Respirology Case Reports* published by John Wiley & Sons Australia, Ltd on behalf of The Asian Pacific Society of Respiriology.

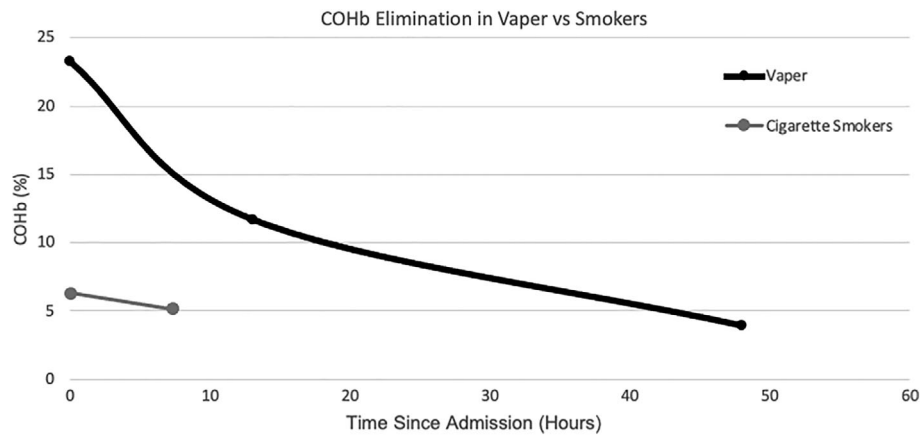


FIGURE 1 Carboxyhaemoglobin (COHb) level (%) in cigarette smokers compared to a vaper at the time of, and post, admission, noting that the normal COHb range is 3%–5%. Data from two of the vaper's admissions have been removed due to being incomplete

returned to normal levels within an average of 47 h using targeted oxygen therapy, non-invasive ventilation and standard treatment directed towards infective exacerbations of COPD. She also received chest physiotherapy and smoking cessation education during her admissions.

Given our patient's elevated COHb, her home was assessed for sources of carbon monoxide (CO) accumulation on two occasions by qualified gas fitters and her husband underwent ABG assessment. All testing was normal, although what is thought to have been a flue gas exhaust pipe was replaced as a precautionary measure following one of her initial admissions. Furthermore, she was not known to be exposed to automobile-related CO.

On her final admission, she had an asystolic collapse at home and could not be resuscitated.

DISCUSSION

This case highlights two important observations. First, that the combination of vaping and cigarette smoking, or vaping alone was associated with nearly four times greater COHb levels than are seen with cigarette smoking. Second, such abnormal COHb levels caused acute-on-chronic high-output right heart failure.

At the time of each admission, our patient presented with signs and symptoms of CO poisoning that were in keeping with the severity of her admission COHb levels, which averaged 23.3% (Figure 1). Her average COHb level was notably higher than that of 12 other smokers from our hospital who had average COHb levels of 6.3% on admission, ranging from 3.1% to 11.1%. The latter group of patients' COHb levels were within the expected range of 3%–15% for cigarette smokers.¹ However, it should be noted that two previous case reports found COHb levels of up to 26.7% in a smoker with a 38 pack-year history, and 24.2% in a 2 pack per day smoker, respectively.²

Given the majority of our patient's presentations were during the years that she was exposed to both vape-related aerosol and cigarette smoke, either directly or indirectly, and her COHb levels normalized during admissions which was a time when she was not exposed to either, the possibility of her vaping device being the source of her CO poisoning is raised. In the literature, we found two papers suggesting that certain vaping devices, power settings and e-fluids may increase CO emissions, with top coil vaping devices potentially posing greater harm than cigarette smoking given their higher levels of CO output.^{3,4} Given the strength of our patient's e-fluid, it is likely that she was using a high-powered device, therefore placing her at greater risk of cardiovascular disease as a result of increased CO exposure.⁴

During the series of admissions, our patient developed pulmonary arterial hypertension. Underlying lung disease and obstructive sleep apnoea can result in this condition; however, in this case, there was a higher cardiac output than would be expected which we believe that caused high-output right heart failure. Although acute high-level CO poisoning can lead to tachycardia and left ventricular heart failure due to direct myocardial toxicity,⁵ we do not think this occurred in our patient as pulmonary capillary wedge pressures were normal during several right heart catheterizations. Research on chronic CO exposure is scant; however, one early study looking at chronic COHb levels above 16% showed resultant tachycardia and an increased cardiac output.⁶ In our patient's case, we believe that vaping either alone or in combination with cigarette smoking led to high levels of chronic CO exposure which together with other risk factors, caused progressive high-output right ventricular failure and preservation of left ventricular function until late in her disease course.

The 2021 update to smoking cessation guidelines by the Royal Australian College of General Practitioner's states that nicotine-containing vaping products may be a reasonable intervention if smoking cessation has failed with first-line therapy.⁷ Therefore, the safety of vaping devices and e-fluid

as well as chronic CO exposure and its effect on the cardio-respiratory system should be further explored.

In summary, this case highlights exceedingly high COHb levels which (a) we attribute to either vaping alone or in combination with cigarette smoking and (b) caused high cardiac output pulmonary arterial hypertension with resultant right heart failure.

ACKNOWLEDGMENTS

The authors thank the Alfred Health Respiratory, Emergency and Intensive Care Department doctors, nurses and allied health staff for their efforts in managing our patient with great expertise and care.

CONFLICT OF INTEREST

None declared.

AUTHOR CONTRIBUTION

Audrey K. Grech, Matthew T. Naughton and Dominic T. Keating treated the patient. Audrey K. Grech wrote the patient presentation details and created the figure. Daniel J. Garner collected data of cigarette smokers. Dominic T. Keating and Matthew T. Naughton provided specialist opinion on the patient's presentation. All authors performed the literature research and corrected the manuscript in a substantial way.

DATA AVAILABILITY STATEMENT

Patient data are available on hospital medical records.

ETHICS STATEMENT

The authors declare that appropriate written informed consent was obtained for the publication of this manuscript and accompanying images.

ORCID

Matthew T. Naughton  <https://orcid.org/0000-0003-2734-0841>

REFERENCES

1. Ernst A, Zibrak JD. Carbon monoxide poisoning. *N Engl J Med*. 1998; 339(22):1603–8.
2. Duran C, Winnicka L, Subramanian CR, Devine S. Recurrent carbon monoxide poisoning due to smoking leading to cognitive decline. *Chest J*. 2016;150(40):423A.
3. Son Y, Bhattarai C, Samburova V, Khlystov A. Carbonyls and carbon monoxide emissions from electronic cigarettes affected by device type and use patterns. *Int J Environ Res Public Health*. 2020;17(8):2767.
4. El-Hellani A, Al-Moussawi S, El-Hage R, Talih S, Salman R, Shihadeh A, et al. Carbon monoxide and small hydrocarbon emissions from sub-ohm electronic cigarettes. *Chem Res Toxicol*. 2019;32(2): 312–7.
5. Lampropoulos CEE, Sklavou C, Anastogiannis V, Papanikolaou V, Tsilivarakis D, Vasiliou V. Acute heart failure in a healthy young patient after severe carbon monoxide poisoning. *Eur J Case Rep Intern Med*. 2019;6(12):001340.
6. Chiodi HP, Dill DB, Consolazio F, Horvath SM. Respiratory and circulatory responses to acute carbon monoxide poisoning. *Am J Physiol*. 1941;134:683–93.
7. Royal Australian College of General Practitioners. *Pharmacotherapy for smoking cessation: a guide for health professionals*. Sydney: RACGP; 2021.p. 31–53.

How to cite this article: Grech AK, Keating DT, Garner DJ, Naughton MT. A case of extreme carboxyhaemoglobinemia due to vaping. *Respirology Case Reports*. 2022;10:e0942. <https://doi.org/10.1002/rcr2.942>