IMAGE CHALLENGE

The blue patient

CLINICAL INTRODUCTION

A 46-year-old man was brought to the ED after being found in an enclosed sauna minimally responsive, clammy and confused.

On arrival he was alert, smiling and recalled sniffing a 'herbal' bottle followed by nausea, dizziness, poor balance, inability to stand up, shortness of breath, palpitations, sweating and confusion. He had a past history of back pain.

He had blue-purplish skin with central and peripheral cyanosis (figures 1 and 2). His observations were: HR 134 bpm, BP 108/68 mm Hg, RR 32/min, temperature 36.3°C and blood glucose 7.2 mmol/L.

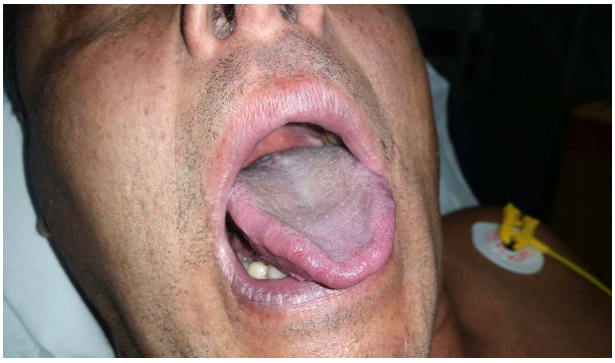

Figure 1 Facial and oral colour on presentation.

Figure 2 Hand colour on presentation.

Pulse-oximeter saturation was 85% on air that only improved to 93% on 15 L/min oxygenation. An arterial blood sample was dark coloured.

QUESTION

What is the diagnosis?

A. Carbon monoxide poisoning
B. Congenital heart disease
C. Nitrite-induced methaemoglobinaemia
D. Cyanide poisoning

For the answer see page 896

The blue patient

From the question on page 890

ANSWER: C

Nitrite-induced methaemoglobinaemia

Oxygenated ABG: methaemoglobin 39%, carboxyhaemoglobin 0.5%, pO_2 36.2 kPa, pCO_2 4.76 kPa, pH 7.36, BE −4.7 mmol/L, bicarbonate 20.8 mmol/L, lactate 3.8 mmol/L.

Alkyl nitrite-induced methaemoglobinaemia due to sniffing of 'popper' bottles was diagnosed.

Methaemoglobin is oxidised deoxyhaemoglobin with its haem iron in the Fe^{3+} state. Cytochrome-b5-reductase is the primary enzyme maintaining methaemoglobin levels at ≤1% of total haemoglobin. Methaemoglobinaemia is commonly acquired from potent xenobiotics such as nitrites, nitrates, chlorates and quinones. Congenital methaemoglobinaemia is rare.[1] Methaemoglobin is deeply pigmented, causing central and peripheral cyanosis with chocolate-brown-coloured blood at levels of 15%–20%. Increasing levels (20%–50%) result in dizziness, weakness, headache, dyspnoea and syncope. Severe poisoning (50%–70%) causes tachypnoea, stupor, coma, dysrhythmias, acidosis and seizures. At >70% methaemoglobinaemia can be fatal.[1 2]

The effects of methaemoglobinaemia can be confused with those of carbon monoxide or cyanide. However, carbon monoxide and cyanide-poisoned patients rarely present cyanotic. Carbon monoxide poisoning usually causes pallor except for excessive exposures when, similar to cyanide poisoning, a cherry-red skin colour can occur. Both poisonings usually cause bright-red-coloured blood and normal pulse-oximeter oxygen-saturation readings. Conventional pulse oximeters, unlike pulse CO-oximeters, do not differentiate carboxyhaemoglobin from oxyhaemoglobin resulting in falsely elevated oxygen saturations. Cyanide poisoning causes tissue hypoxia with normal arterial oxygen tension and narrow arterial–venous oxygen difference. The patient's age, past medical history and lack of heart murmurs excluded cyanotic congenital heart disease, a condition that, with decompensation, can present in similar fashion.[1]

Treatment is usually advised if symptomatic or when the methaemoglobin level is >25%. Methylene blue (1–2 mg/kg) acts within the erythrocytes to form leucomethylene blue, a reducing agent of oxidised haemoglobin. Methylene blue can cause local burning pain on injection and interferes with pulse-oximeter readings resulting in falsely depressed oxygen saturation values. Monitoring is advised because methaemoglobinaemia can rebound after effective therapy.[1–3]

The patient was treated with methylene blue 2 mg/kg intravenously. Pulse-oximeter saturations dropped to 70% during infusion. The patient became even bluer in colour due to the methylene blue itself (figure 3A). Pulse-oximeter reading was 99% 5 min after the completion of infusion. The patient's colour turned pink and he felt better (figure 3B). His arterial blood turned red from chocolate brown.

Repeat ABG showed methaemoglobin 1.5% and lactate 1.7 mmol/L. The patient remained asymptomatic during overnight observation. Following treatment, his urine turned blue.

Adrian Spiteri

Correspondence to Dr Adrian Spiteri, Emergency Department, St George's University Hospitals NHS Foundation Trust, Blackshaw Road, London SW17 0QT, UK; spiteri.adrian@gmail.com

Competing interests None declared.

Patient consent Obtained.

Provenance and peer review Not commissioned; externally peer reviewed.

Data sharing statement I allow data sharing of the original article as needed by the *EMJ*. The data for this image challenge is available to me after looking after the patient personally and obtaining consent to publish the case from the patient. There is additional unpublished information of the case in my possession that has not been included due to word count restrictions.

To cite Spiteri A. *Emerg Med J* 2016;**33**:896.

Accepted 27 March 2016

Emerg Med J 2016;**33**:896. doi:10.1136/emermed-2016-205729

REFERENCES

1 Hoffman RS, Howland MA, Lewin NA, *et al*. *Goldfrank's toxicologic emergencies*. 10th edn, McGraw-Hill Education / Medical, 2015.
2 do Nascimento TS, Pereira RO, de Mello HL, *et al*. Methemoglobinemia: from diagnosis to treatment. *Rev Bras Anestesiol* 2008;58:651–64.
3 Clifton J II, Leikin JB. Methylene blue. *Am J Ther* 2003;10:289–91.

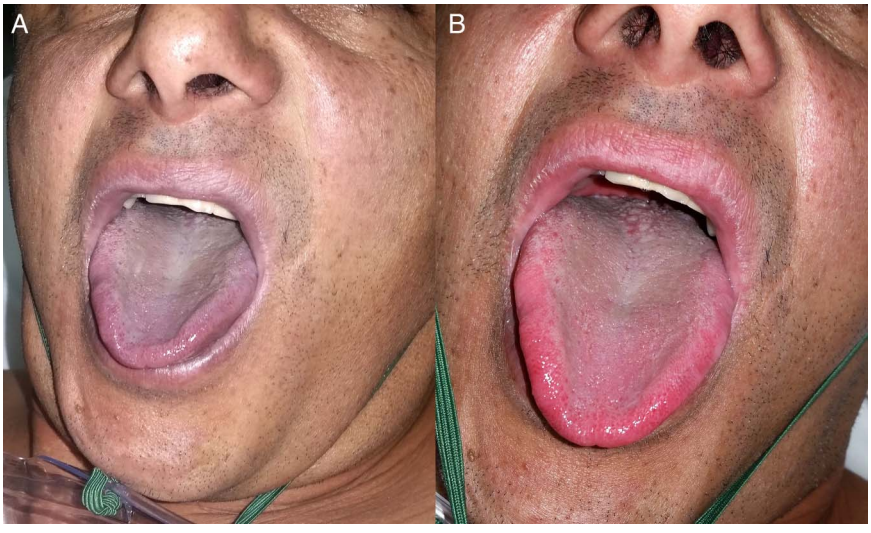

Figure 3 (A) Facial and oral colour during methylene blue treatment. (B) Facial and oral colour after treatment.