The pitfalls of oximetry

Introduction

Pulse oximetry is widely used in the clinical assessment of oxygen saturations and to assess respiratory function. The oximeter detects light absorption at two different wavelengths of light and uses this to calculate the amount of oxyhaemoglobin and deoxyhaemoglobin present. Haemoglobin variants can result in additional absorption in these spectra leading to inaccurate oxygen saturations.

Clinicians rely on oximetry for non-invasive assessment of oxygen levels. This article presents a case of an unusual haemoglobinopathy causing falsely low oxygen saturations and reviews the pitfalls and limitations of pulse oximetry.

Discussion

The causes of hypoxaemia can be divided into five categories which are outlined in *Table 1*.

The normal imaging and pulmonary function excluded many of the causes of hypoxia. The sleep study in isolation would suggest that the patient was hypoventilating but she had neither the clinical symptoms of hypoventilation (e.g. headaches, sleepiness, snoring or witnessed apnoeas) nor an underlying clinical diagnosis to precipitate hypoventilation.

The intermittent episodes of shortness of breath are felt to be panic attacks and patient has remained well after 1 year of follow up.

Table 1. Differential diagnosis of hypoxaemia

Low inspired oxygen	Decreased inspired oxygen concentration (e.g. altitude)
Hypoventilation	Obesity hypoventilation syndrome
	Opioid toxicity
Right-to-left shunting	Tetralogy of Fallot
	Intrapulmonary arteriovenous malformations
Ventilation and perfusion mismatch	Pulmonary emboli
	Emphysema
Diffusion defects	Interstitial lung disease

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Haemoglobin lansing

More than 1000 haemoglobin variants have been identified (Hardison et al, 2002). The majority do not interfere with oximetry readings. Haemoglobin lansing was first described in 2009 (Sarikonda et al, 2009). It is the consequence of a single point mutation (CAC to CAG) at codon 87 of the alpha-2 gene. Oxygen affinity is normal and thus it is of no clinical significance.

Pulse oximeters emit light at wavelengths of 660 nm and 940 nm and measure the differential absorption of oxyhaemoglobin and deoxyhaemoglobin at each of these wavelengths. It is thought that haemoglobin lansing (Zur et al, 2008) has spectral absorption at 660 nm meaning that the oximeter mistakes it for deoxyhaemoglobin, leading to a spuriously low oximeter reading.

Arterial sampling and use of blood co-oximetry is more accurate as modern co-oximeters measure absorbance at over 100 wavelengths on a spectrum from 450–700 nm (Pamidi et al, 2009).

This case highlights the importance of understanding the limitations of pulse oximetry. When a spurious reading is considered this can be confirmed by arterial blood gas sampling. A discrepancy between oxygen saturations and peripheral capillary oxygen saturation should lead the clinician to consider investigation for rare variant haemoglobins. BJHM

LEARNING POINTS

- Knowledge of the mechanism of action and limitations of pulse oximetry enables spurious results (when present) to be suspected.
- In the event of a suspected spurious reading from a pulse oximeter clinicians should have a low threshold for performing arterial blood gas sampling.
- Patients should not be diagnosed as having nocturnal hypoventilation unless they have an underlying reason to explain their hypoventilation (e.g. restrictive lung disease, neuromuscular disease).

Case Report

A 38-year-old patient of Caribbean descent with a history of epilepsy was seen at the respiratory outpatient department. During an inpatient stay, her oxygen saturations on pulse oximetry (SpO_2) had been noted to be 91% on ambient air.

She reported variable shortness of breath. She had no symptoms of sleep-disordered breathing. She was an ex-smoker with a 10-pack year history and her medications were levetiracetam, clobazam, pantoprazole, folic acid and calcium supplements. Her SpO_2 was confirmed as 90% on room air. Clinical examination was unremarkable.

Pulmonary function testing revealed forced expiratory volume in 1 second (FEV_1) 1.97 litres (86% predicted), forced vital capacity (FVC) 2.49 litres (94% predicted), and FEV_1/FVC ratio of 79%. Gas transfer coefficient for carbon monoxide was 6.94 mmol/min/kPa (86% predicted). A chest radiograph, computed tomography of the chest and echocardiogram with bubble study were all normal. A sleep study showed mean saturations of 88% with a normal 4% oxygen desaturation index.

Given the lack of symptoms of hypoventilation and normal investigations the possibility of a spurious recording from the pulse oximeter was considered. An arterial blood gas on room air revealed pH 7.43, pO_2 12.8 kPa, pCO_2 4.93 kPa. Arterial blood oxygen saturation was 97%, methaemoglobin level was 0.8% (normal range 0–2%). Sickle cell screening and routine haemoglobinopathy panels were negative.

She was referred to the haematology department for further investigation and DNA sequencing confirmed the presence of haemoglobin lansing.

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Periorbital subcutaneous emphysema: an unreported presentation of perforated duodenal ulcer

The incidence of peptic ulcer disease has declined steadily in recent years owing to the wider availability of proton-pump inhibitors; despite this, complications are common (Hermansson et al, 2009). Perforated duodenal ulcers are a recognized serious complication of peptic ulcer disease, typically presenting with abdominal pain and haemodynamic shock (Thorsen et al, 2013). Facial subcutaneous emphysema usually indicates injury to the upper aerodigestive tract (Wang et al, 2004).

A 71-year-old man presented with a 2-day history of epigastric pain and bilious vomiting with respiratory and haemodynamic compromise. He had bilateral periorbital subcutaneous emphysema (*Figure 1*). A computed tomography scan confirmed the presence of pneumomediastinum and bilateral pneumothoraces as a consequence of a perforated duodenal ulcer (*Figure 2*).

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The presence of subcutaneous emphysema can indicate an occult injury in the gastrointestinal tract. A thorough clinical examination to identify the distribution of subcutaneous emphysema can provide clues to the location of perforation (Oetting et al, 1955). Subcutaneous emphysema of the face can indicate a perforated duodenal ulcer. **BJHM**

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Figure 1. Extensive subcutaneous emphysema still persisting around both eyes 10 days post-admission.

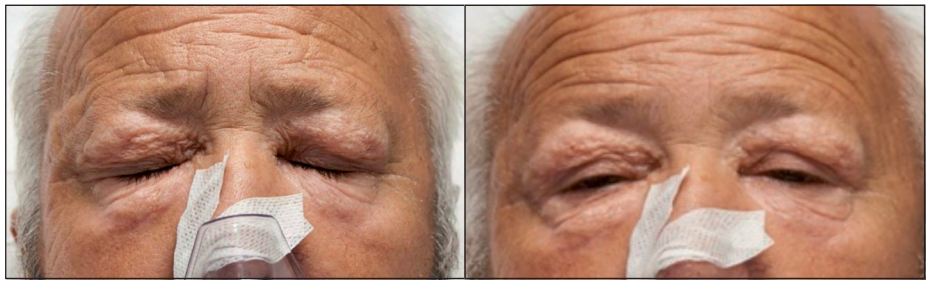

Figure 2. a. Axial computed tomography scan on lung window settings showing extensive air tracking along the tissue planes including the mediastinal structures, and a left pneumothorax. b. Coronal reformatted computed tomography scan on lung window settings.