Dark green blood in the operating theatre

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In October, 2005, a 42-year-old man presented to our hospital, having developed a compartment syndrome in both lower legs. He was a smoker; his medical history included migraine. His regular medications were sumatriptan, which he had been taking at a dose of 200 mg per day, for several months, diclofenac, and zopiclone.

Urgent fasciotomies were required. Preoperative physical examination revealed an alert white Canadian, with a rather dark complexion. His heart rate, blood pressure, oxygen saturation (SpO_2) and respiratory rate were 110 beats per min, 135/80 mm Hg, 91% on ambient air, and 16 breaths per min respectively. His only abnormal blood result was a creatine kinase concentration of 43 384 U/L. A toxicology screen was negative. The patient was given intravenous morphine, dimenhydrinate, and sodium bicarbonate, and general anaesthesia was induced uneventfully. However, several attempts to insert an indwelling radial-arterial catheter yielded dark, greenish-black blood. Eventually, the catheter was advanced, and pressure transduction confirmed arterial placement. The displayed SpO_2 was 96% on supplemental oxygen (inspired oxygen fraction [F_iO_2], 0.5). A sample of the greenish-black blood was sent to the laboratory for co-oximetry. The oxygen saturation (SaO_2) was 94%, and the oxygen partial pressure (PaO_2) 135 mm Hg. The methaemoglobin concentration was within the normal range, at 1·17 g/L (0·9%), as was the carboxyhaemoglobin concentation—but the analyzer displayed an alert to the presence of sulfhaemoglobin. Quantitative analysis (Beckman DU65 Spectrophotometer, Beckman Coulter, Mississauga, Canada) revealed a sulfhaemoglobin concentration of 2 g/L. The patient recovered uneventfully, and stopped taking sumatriptan after discharge. When seen 5 weeks after his last dose, he was found to have no sulfhaemoglobin in his blood.

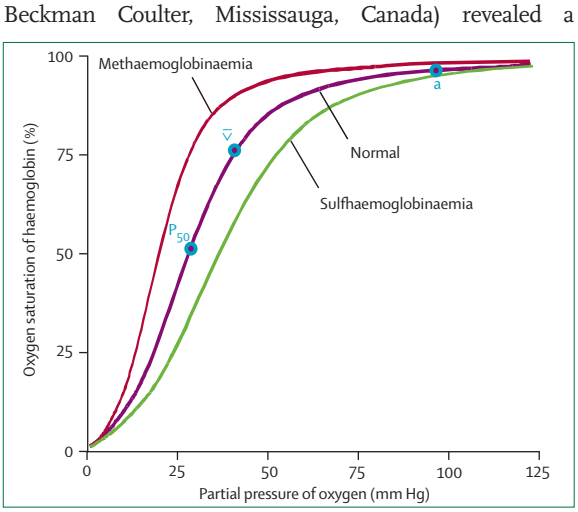

Figure: **Effects of sulfhaemoglobinaemia and methaemoglobinaemia on the oxyhaemoglobin dissociation curve**
Unlike methaemoglobinaemia, sulfhaemoglobinaemia shifts the oxyhaemoglobin dissociation curve to the right, promoting oxygen delivery from haemoglobin to the tissues. To an extent, this counteracts the deleterious effects of sulfhaemoglobinaemia on oxygen transport. P_{50}=partial pressure of oxygen at which haemoglobin is 50% saturated with oxygen; a=normal arterial partial pressure; v̄=normal mixed venous partial pressure.

Cyanosis is usually caused by deoxyhaemoglobin in the capillaries imparting a blue colour to the skin and mucous membranes. This requires an arterial deoxyhaemoglobin concentration of at least 25–50 g/L: cyanosis can therefore be absent in anaemia even when a high proportion of haemoglobin is deoxygenated. Increased arterial deoxyhaemoglobin concentrations can be caused by lung disease, or by blood being shunted from the venous to the arterial circulation without passing through the lungs, as happens in certain congenital cardiovascular malformations. Occasionally, however, cyanosis is caused not by deoxyhaemoglobin but by methaemoglobin or, more rarely, sulfhaemoglobin. Methaemoglobin is formed when haemoglobin is oxidised from $HbFe^{2+}$ to $HbFe^{3+}$. It constitutes up to 2% of haemoglobin even in healthy people; higher concentrations are usually caused by adverse drug reactions. Sulfhaemoglobin is formed when a sulphur atom is incorporated into the porphyrin ring of the haem group.[1] The formation of sulfhaemoglobin can be caused by medications, including sulfonamides.[2] It is possible that our patient's arguably excessive intake of sumatriptan, which contains a sulfonamide group,[3] caused his sulfhaemoglobinaemia. Although they affect the behaviour of normal haemoglobin (figure), neither methaemoglobin nor sulfhaemoglobin transport oxygen. Dyshaemoglobinaemias should be considered as possible causes of cyanosis, particularly in the presence of a relatively normal PaO_2. It has previously been reported that an arterial sulfhaemoglobin concentration of 5 g/L produces detectable cyanosis;[3] but we observed a dark complexion, and discoloured blood, at 2 g/L. Notably, not all co-oximeters are capable of detecting sulfhaemoglobin; moreover, because sulfhaemoglobin has a peak absorption at 620 nm, sulfhaemoglobinaemia can produce erroneous readings on conventional pulse oximetry, which uses wavelengths of 660 and 940 nm.[4,5] Sulfhaemoglobinaemia generally resolves with erythrocyte turnover; however, transfusion can be necessary in severe cases.

References

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