

Case Report of Methaemoglobinaemia

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ABSTRACT

A 70 year old gentleman who was admitted to hospital with flu-like symptoms, and later confirmed to be influenza A positive. His past medical history included Hairy Cell Leukaemia (HCL), hyperlipidaemia and hypertension. On review the patient was requiring 15L of oxygen via a non rebreathe mask, and despite this was still hypoxic. His ABG showed a significant discrepancy between SaO₂ and PaO₂ and an elevated methaemoglobin (MetHb) %. This was secondary to dapsone and was treated with Methylene Blue (MB) to good effect. This case report details this unusual condition in more detail.

Keywords: Methaemoglobinaemia; Hairy Cell Leukaemia (HCL); Hyperlipidaemia; Hypertension

BACKGROUND

Methaemoglobinaemia is a rare disorder in which MetHb reaches in excess of 1%. MetHb is haemoglobin in which the iron atom is oxidised from 2⁺ (ferrous) to 3⁺ (ferric). Ferric iron is unable to bind oxygen, resulting in a functional anaemia and impaired tissue oxygenation [1]. Methaemoglobin is formed naturally in the blood, as result of oxidative stress, however in health levels are maintained at <1% by anti-oxidant pathways. The most important of these are the NADH-dependent cytochrome b5 reductase (methemoglobin reductase) pathway and the NADPH-dependant methemoglobin reductase pathway [2].

Elevated MetHb occurs either when there are defects in these protective enzyme systems (congenital, rare -e.g. Glucose-6-phosphate deficiency, cytochrome b5 reductase deficiency) or when these systems are overwhelmed by powerful oxidising substances (acquired, more common). Acquired causes include numerous drugs, particularly sulfonamides (dapsone, sulfasalazine), rasburicase, metoclopramide, local anaesthetics, aniline dyes and nitrites (NO, 'poppers'). Levels of >15% can cause cardiac (angina, dyspnoea) & neurological (confusion, reduced GCS) symptoms, and levels >70% are generally deemed to be fatal.

CASE PRESENTATION

This 70 year old gentleman presented to his local ED complaining of a 9 day history of cough productive of white sputum and reduced exercise tolerance. He had recently been on holiday to the Mediterranean. Otherwise the patient was well and on initial assessment appeared stable. He had no chest pain.

Past medical history was significant for HCL, treated with cladribine in 2011, hypertension, hyperlipidaemia and glaucoma. His drug history consisted of losartan, aspirin, latanoprost, dapsone and aciclovir. He was a non-smoker & occasionally consumed alcoholic beverages. His usual exercise tolerance was essentially unlimited.

INVESTIGATIONS

Admitting observations were as follows: pulse 112; BP 142/82 respiratory rate of 20; oxygen saturation s of 86% on room air & a temperature of 37°. Initial blood tests revealed a modestly elevated CRP of 86, an eGFR of 47 and a white cell count of 6. His influenza swab was positive for influenza A. D Dimer & troponin levels were normal. He had bloods cultures performed, which were subsequently found to be negative. His chest X ray showed pleural plaques but no new abnormality. ECG showed a sinus tachycardia only.

ABG performed on room air revealed a PAaO₂ of 8.01, however SaO₂ as recorded on the ABG was only 80% -Significantly discrepant. Based on his low SaO₂ he was placed on 15L via non rebreathe mask. Despite this SaO₂ remained low. An ABG was performed which was as follows: Of note there is a significant discrepancy between his PaO₂ and SaO₂, typical of methaemoglobinaemia. His MetHb was significantly elevated at 20.2%.

DIFFERENTIAL DIAGNOSIS

Based on the symptoms he initially presented with, an upper or lower respiratory tract infection was felt to be most likely. His chest had generalized crepitations on auscultations and he appeared stable from the end of the bed. He was however profoundly hypoxic

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according to his SaO_2 , a finding significantly at odds with the rest of his assessment. PE was considered but excluded by a normal D-Dimer, and it would be very unusual to be so hypoxic without having other features of pulmonary embolism. Pneumonia was excluded based on his CXR appearances. He was placed on high flow oxygen but despite this his SaO_2 did not improve. The ABG was key to establishing the diagnosis in this case: The discordant PaO_2 and SaO_2 and significantly elevated MetHb%.

TREATMENT

The patient was started on Oseltamivir and antibiotics to cover for possible superadded bacterial infection. After liaison with haematology and intensive care he was treated with methylene blue, a powerful reducing agent which is able to reduce the iron in MetHb from 3^+ to 2^+ , thus allowing it to transport oxygen once again. His dapsone was permanently ceased.

OUTCOME

This patient made a good recovery & was discharged home after a nine day stay in hospital. He is being followed up in the haematology outpatient clinic.

CONCLUSION

It is important to fully consider all side effects of medications when considering your differentials. This applies to medicines that the patient has been on for a long time. Dapsone has a unique side effect profile & its inclusion in a patient's regular medicine should widen ones differential list considerably. If there is significant discrepancy between 1) How the patient appears from the end of the bed and their oxygen saturations and 2) Their SaO_2 and PaO_2 on ABG always pay attention to the MetHb %. This information is available as routine on an ABG but is often overlooked for the more familiar parameters.

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