Acute Methaemoglobinaemia Secondary to Intentional Dapsone Overdose

Sir

Methaemoglobinaemia is a rare but potentially life-threatening hemoglobinopathy. It is characterized by cyanosis that is not responsive to oxygen therapy. We report a case of acute methaemoglobinaemia secondary to intentional dapsone overdose. A 34 year old lady presented to our institution with a history of sudden onset of dyspnoea. Her level of consciousness had reportedly deteriorated dramatically during ambulance transportation and her Glasgow Coma Scale was 3/15 on arrival. She was normothermic and hemodynamically stable, but had a respiratory rate of 30 breaths per minute and was cyanosed with an SpO\textsubscript{2} of 83% on 15 litres of oxygen via a non-rebreather mask.

She was emergently intubated and her lungs were ventilated with 100% oxygen. There was good air entry bilaterally but she remained cyanosed with an SpO\textsubscript{2} of 83%. Physical examination was otherwise unremarkable. Her chest x-ray and routine blood tests including toxicology screen was normal. Her initial arterial blood gas (ABG) showed a pO\textsubscript{2} of 54.1 kPa, pH of 7.42, and SpO\textsubscript{2} of 100%. Analysis by co-oximeter showed an oxyhemoglobin level of 55% and a methaemoglobin level of 44.4%.

A diagnosis of methaemoglobinaemia was made and methylene blue was administered at a dose of 1.5 mg/kg with immediate improvement of the patient’s colour and a rise in the SpO\textsubscript{2} reading on the monitor to 100%. A number of empty tablet containers were subsequently found at the patients bedside at home. Our patient had a diagnosis of HIV which was treated with antiretrovirals and prophylactic dapsone therapy. She had taken an intentional overdose of dapsone leading to acute methaemoglobinaemia. Her methaemoglobin levels dropped over the following days with ongoing management and she ultimately made a full recovery. Methaemoglobinaemia can occur in either hereditary or acquired forms. Amyl nitrite, aniline dyes, benzocaine, lignocaine, nitroglycerine, phenytoin, prilocaine, primaquine, pyridine, silver nitrate, sulphonamides and dapsone have all been implicated in acquired methaemoglobinaemia.

Methaemoglobinaemia was suspected in our patient in the setting of cyanosis unresponsive to oxygen therapy. Both standard bedside pulse oximetry and blood gas analysis were misleading as is commonly the case in methaemoglobinaemia. Pulse oximetry measures the relative light absorption at the wavelengths of 660 nm and 940 nm to differentiate oxyhaemoglobin and deoxyhaemoglobin. At these wavelengths red and infrared light are absorbed at a 1:1 ratio by methaemoglobin, corresponding to an arterial saturation of 85% by the pulse oximetry conversion algorithm for oxy and deoxyhaemoglobin. Thus with increasing levels of methemoglobin, pulse oximetry readings will plateau at 85% and overestimate the true level of oxygen saturation. Of note there was a large gap between the oxygen saturation measured on pulse oximetry and on ABG analysis in our patient. This prompted an ABG analysis in the co-oximeter which established the diagnosis of methaemoglobinaemia and prompted the successful treatment.

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References

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