Medical Image of the Week: Methemoglobinemia

A 62-year-old man with a sternal infection post-coronary artery bypass grafting was transferred because of increasing oxygen requirements, decreased mental status and *Candida paralopsis* fungemia. He had been treated with multiple antibiotics including sulfonamides and had eventually undergone a tracheostomy. Cetacaine was used for complaints of a sore throat. Physical examination showed cyanotic lips and tongue (Figure 1A). Blood drawn was a chocolate brown color (Figure 2). His SpO2 was 88%, however, arterial blood gases showed his SaO2 was 100% and his PaO2 453 mmHg. Co-oximetry showed 26% methemoglobin. He was administered 0.2mL/kg of 1% solution of methylene blue and his cyanosis rapidly cleared (Figure 1B).

Methemoglobinemia is a condition characterized by increased quantities of hemoglobin with iron oxidized to the ferric (Fe3+) form (1). Methemoglobin is useless as an oxygen carrier and thus causes a varying degree of cyanosis and hypoxia. It can be genetic but is usually caused by exposure to drugs or toxins. Symptoms are proportional to the fraction of methemoglobin. A normal methemoglobin fraction is about 1%. Symptoms associated with higher levels of methemoglobin are: 3-15% - cyanosis; 25-50% - headache, dyspnea; 50-70% - cardiac arrhythmias, altered mental status, delirium, seizures, coma; >70% - death. Drugs most commonly associated with methemoglobinemia include
topical and injected local anesthetics (benzocaine, lidocaine, cetacaine), sulfonamides (dapsone), and nitrates (nitroprusside).

Intravenous methylene blue, a reducing agent, is the traditional antidotal agent (1). Exchange transfusion and hyperbaric oxygen treatment are second-line options for patients with severe methemoglobinemia who do not respond to methylene blue or who cannot be treated with methylene blue (e.g., those with glucose-6-phosphate dehydrogenase [G6PD] deficiency).

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Reference