Anesthesia-induced methemoglobinemia in a patient with Hansen’s Disease: a case report.

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INTRODUCTION

Acquired methemoglobinemia (methga) caused by administration of local anesthetics is an uncommon event, occurring in about 1 in 7000 exposures. Although rare, the consequences of methga can be severe, including seizures, solid organ damage, and even death. In the field of otolaryngology, patients are administered local anesthetics in the clinical and operative settings, increasing the likelihood of developing methga. Thus, familiarity with this condition facilitates prompt recognition and treatment of methga which is critical for good outcomes. This article presents a case of methga during management of an obstructed airway and discusses its physiology, clinical diagnosis, and treatments.

CASE DESCRIPTION

Our patient is a 52-year-old with a past medical history significant for poorly-differentiated squamous cell carcinoma of the left lateral tongue treated with partial glossectomy, left neck dissection, and adjuvant radiation in 2008. She also has a history of hypothyroidism and leprosy (Hansen’s Disease).

Two days prior to presentation to the Head and Neck Clinic, she was taken to the operating room for direct laryngoscopy and biopsy of lesions suspicious for recurrence. In the operating room, she was topically anesthetized with 4 ml of 4% lidocaine via nebulizer. Two sprays of cetacaine (14% benzocaine, 2% butyl aminobenzoate, 2% tetracaine) were applied to the pharyngeal mucosa. She received fentanyl, propofol, rocuronium, lidocaine (3mg) intravenously after the airway was secured. She was maintained on inhaled isoflurane. After the procedure, her vital signs remained stable while in the recovery room, and she was discharged home the day of surgery.

PHYSIOLOGY OF METHEMOGLOBINEMIA

Hemoglobin is a tetrameric protein consisting of four heme-protoporphyrin rings. Each of the four heme-protoporphyrin rings contains an iron atom that reversibly binds oxygen for transport and delivery to tissues. The iron atom can exist in a reduced, ferrous (2+) form or an oxidized, ferric (3+) form. The ferrous atom is oxidized to the ferric state when binding an oxygen molecule; however, it is reduced when oxygen is unloaded. Oxidative events during metabolism can oxidize the ferrous form of hemoglobin. When hemoglobin is oxidized to the ferric state without the binding of oxygen, then it becomes methemoglobin (metHb). MetHb binds water instead of oxygen, causing a leftward shift in the oxyhemoglobin dissociation curve. This leads to a reduction in oxygen carrying capacity and decreased oxygen unloading of the remaining ferrous molecules. Fortunately, homeostatic mechanisms exist to counteract this natural production of metHb, which peaks at 2% of the total amount of hemoglobin. The major pathway involves nicotinamide adenine dinucleotide (NADH) cytochrome-b5 reductase facilitating transfer of an electron to metHb (Figure 1). In the minor pathway, nicotinamide adenine dinucleotide phosphate (NADPH) cytochrome-b5 reductase catalyzes an electron transfer from NADPH to metHb. This reaction is significantly accelerated with a cofactor such as methylene blue.

CONCLUSION

This case highlights the increased potential for developing methemoglobinemia with lidocaine while on concomitant oxidizers, like dapsone. Early recognition of the causes, diagnosis, and treatment of methemoglobinemia is essential for a good outcome.

REFERENCES