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EMLA-induced methemoglobinemia after laser-assisted hair removal procedure

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ABSTRACT

This is the case of a 23-year-old female with a past medical history of ADHD and Depression who was evaluated in the emergency department for perioral cyanosis and hypoxia after application of the eutectic mixture of lidocaine and prilocaine (EMLA) local anesthetic prior to a laser-assisted hair removal procedure. This report illustrates a case of methemoglobinemia which is a rare but significant complication of topical anesthetic use.

1. Introduction

Topical anesthetics, which work by reversibly inhibiting the transmission of nerve impulses, are commonly utilized for pain reduction in a variety of medical settings [1,2]. Despite their widespread use, awareness of their potential systemic toxicities are not well recognized [3]. The eutectic mixture of 2.5% lidocaine and 2.5% prilocaine (EMLA cream) is a commonly utilized form of local anesthetic. Despite limited systemic absorption from its topical application, systemic toxicities including methemoglobinemia, CNS, respiratory and cardiovascular effects have been reported [4,5]. Here, we describe the case of a patient who developed methemoglobinemia after application of EMLA cream prior to a laser-assisted hair removal procedure.

2. Case presentation

The patient is a 23-year-old female (65 kg), on Adderall and Bupropion, who was transferred from an urgent care center for perioral cyanosis and lightheadedness after undergoing a laser-assisted hair removal procedure. Three hours prior to the procedure (6 h prior to ED presentation), a total of 150 g of EMLA cream (5 tubes, 30 g per tube) was applied to the patient's torso. Prior to onset of procedure, excess EMLA cream was removed. Approximately 5 h after initial application of EMLA cream, the patient developed palpitations, lightheadedness and perioral cyanosis, prompting her to go to an urgent care center that subsequently referred the patient to the emergency department (ED). Upon arrival to the ED, the initial vital signs were: temperature 36.7 °C (98.2 °F), heart rate 108 beats per minute, blood pressure 116/72 mm Hg, respiratory rate of 18 breaths per minute. The oxygen saturation by pulse oximetry was 88% on room air. On administration of 15 L/min oxygen by nonrebreather mask, the oxygen saturation remained in the mid 80s by pulse oximeter.

On exam, there was perioral cyanosis. Lungs were clear bilaterally, and heart sounds were normal. Neurological examination was grossly intact, including mental status examination. A 12-lead electrocardiogram revealed normal sinus rhythm without any abnormalities. A methemoglobin level obtained from blood assay was 21.8%. Poison control was consulted, and the patient received 65 mg of methylene blue solution intravenously over 30 min (1 mg/kg in 50 mL D5W IVPB) with subsequent improvement of oxygen saturation to 94% on room air after completion of treatment. The patient's perioral cyanosis and palpitations improved, and the patient was discharged over 6 h after initial presentation.

3. Discussion and conclusion

Methemoglobinemia is a condition that occurs when there are increased levels of oxidized hemoglobin in the blood [6,7]. Oxidation of oxygen occurs regularly due to normal metabolic activity of the body, but can be enhanced in the presence of exogenous agents

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with oxidant properties. Oxidized hemoglobin cannot bind oxygen in the blood and causes a functional anemia [8].

Patients with methemoglobinemia experience acute impairment of oxygen delivery to tissues. Severity of symptoms is related to the percentage of met-Hb in the blood. Clinical suspicion of the diagnosis is increased with the presentation of hypoxia that does not improve with oxygen. Methemoglobin interferes with light absorbance at both wavelengths read by traditional pulse oximeters, disrupting saturation readings. Co-oximetry can overcome this limitation by measuring light absorbances at multiple wavelengths [9]. The first step in treatment involves removal of the offending agent, and supportive treatment such as administration of oxygen. Poison control should be contacted. Patients that are symptomatic should additionally be administered methylene blue, which works by providing an artificial electron transporter for the reduction of methemoglobin in the red blood cell [10].

Both lidocaine and prilocaine are capable of generating methemoglobin [5]. Nevertheless, this EMLA-associated toxicity is uncommon due to the relatively limited systemic absorption of this agent [11]. The potential for toxicity is increased with application excessive of the recommended dose and recommended surface area (60 g to 400 cm squared of skin) [12]. Medications that interfere with cytochrome P450 may also lead to increased methemoglobin formation. All of these factors were potential contributors to the toxicity observed in our case.

The exact prevalence of EMLA-induced methemoglobinemia is unknown. A literature search has revealed at least nine cases of EMLA-associated poisonings directly linked to laser-assisted hair removal procedures [5]. Other reports exist describing toxicities associated to EMLA use prior to other procedures such as circumcisions, laceration repairs and IV insertions [13-15]. As EMLA cream is increasingly utilized as an analgesic agent in a variety of settings, it is important to be aware of its potential toxicities. In this report, we describe another case of EMLA-induced methemoglobinemia prior to laser-assisted hair removal, adding to the growing number of reports describing systemic toxicities in association to this analgesic agent.

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