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MINI REVIEW

Cardiac Anesthesia Management of an Acquired Methemoglobinemia Patient

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Abstract

Acquired methemoglobinemia occurs most commonly from the ingestion of medications or toxins that oxidize the ferrous iron of hemoglobin. Anesthetizing patients with methemoglobinemia is a highly specialized task. Nevertheless, cardiac anesthesia for these patients requires high attention on maintenance of precise O_2 delivery to tissues. We herein, describe an uneventful course of an acquired methemoglobinemia patient having on pump mitral valve replacement and two coronary vessel bypass grafting surgery with review of the literature.

Keywords

Methemoglobinemia, Oxygen, Cardiac anesthesia

Abbreviations

ECG: Electrocardiogram; SpO_2: Pulse Oximetry; ABG: Arterial Blood Gas; Hb: Hemoglobin;

GTN: Glyceryl Trinitrate Intensive; ICU: Care Unit; Methb: Methemoglobin; IV: Intravenously

Introduction

Hemoglobinopathies, may be presented to the anesthesiologist as a problem arising from the disease itself, as the primary cause of a surgical procedure, or as an incidental complicating problem during surgery. Methemoglobinemia is rare and difficult to diagnose, because its clinical picture ranges from asymptomatic underdiagnosed cases to potentially fatal cases [1]. Thus, many of these patients may undergo cardiac surgery. In addition to the physiologic goals for the patient, hypothermic cardiopulmonary bypass, low flow states, circulatory arrest, and aortic cross-clamping together with the medications challange the appropriate perioperative care of these patients.

By this case, we review the literature on anesthetic considerations for methemoglobinemia and cardiac surgery.

Case Description

We present a case of a 67-years-old woman (64.5 kg, and 167 cm) scheduled for mitral valve replacement and coronary artery bypass graft surgery. There were no abnormalities on electrocardiogram (ECG), chest radiography, echocardiography and pulmonary function test. During routine physical activities, such as walking and climbing stairs, the patient complained of dyspnea and palpitation. The patient had been previously diagnosed with hypertension, coronary artery, rheumatic heart disease and osteoporosis. In her past history, she had mitral valvuloplasty performed 30 years ago. She called attention to the cardiac arrest she had due to hemodynamic instability caused by severe hypoxia during mitral valvuloplasty. Later she had been explained to have methemoglobinemia and been informed to avoid a list of drugs by her anesthesiologist. On physical examination she was unremarkable. In the

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operating room, standard monitoring was applied (5 lead ECG, non-invasive blood pressure, pulse oximetry - SpO_2). Her SpO_2 was 95%. Intravenous and radial artery catheters were inserted before anesthesia induction. After 3 minutes of preoxygenation, her SpO_2 increased to 99%. Arterial blood gas (ABG) measurement was taken immediately, and it showed; pH: 7.40, PaO_2: 309 mmHg, hemoglobin (Hb): 10.5 g/dL, and metHb rate: 1.2%. This methemoglobinemia was well tolerated before and during anesthesia. In the light of preoperative patient interview, we decided not to use lidocaine at induction. The patient underwent an uneventful induction of anesthesia with 500 mcg fentanyl, 6 mg midazolam, and 10 mg vecuronium. She was intubated with a 7.5 endotracheal tube and placed on inhaled desflurane for maintenance. Intraoperatively, glyceryl trinitrate (GTN) which we used to infuse for hemodynamic control in our cardiac anesthesia practice wasn't given. Instead of GTN, we preferred to have infusion of *esmolol* hydrochloride (Brevibloc®), so as to keep mean arterial pressure ≥ 65 mmHg. As we avoided any oxidizing agent throughout the surgery, we were not suspicious of insufficient tissue oxygenation. We had mild-moderate hypothermia (32 °C), normal hematocrit at 25-27%, pH at 7.4 and high flows like 3.0-3.5 l/min/m^2. The patient underwent a successful mitral valve replacement with two coronary vessels anastomosis. There was no intraoperative evidence of hypoxia or cyanosis. The urine output was greater than 1 ml/kg/hour and appeared clear. No additional blood products were given on cardiopulmonary bypass. The total cardiopulmonary bypass time was 140 minutes and cross-clamp time was 85 minutes. She separated from cardiopulmonary bypass pump with the use of low doses of dobutamine and noradrenaline. At the end of bypass period, a repeat arterial blood gas sample showed that her methemoglobin level had increased from 1.2-3.5%. After surgery, the intubated patient was transferred to the cardiovascular surgery intensive care unit (ICU). She was extubated the next morning uneventfully. She was discharged from the ICU on day 4 and from hospital on day 10 without any complication.

Discussion

Methemoglobin (MetHb) is a kind of hemoglobin (Hb) molecule, that is produced by the oxidation of iron in the hemoglobin, from ferrous state (Fe^{2+}) to ferric state (Fe^{3+}). This results in inability of Hb molecule to bind oxygen and, furthermore, causes a left shift of the oxyhemoglobin dissociation curve [1]. Thus methemoglobin, depending on its level, can lead to cellular hypoxia and, ultimately, complications such as dyspnea, headache, heart failure, and even death [2].

In clinical practice, medications such as local anesthetics (benzocaine and procaine), antibiotics (Dapsone), and nitrites (nitroglycerin/nitric oxide) are the most common causes of MetHb. This is acquired methemoglobinemia which is more frequent than congenital methemoglobinemia. Congenital methemoglobinemia occurs in patients with cytochrome b5 reductase deficiency or hemoglobin M (Hb M) disease [3]. Nevertheless, Methemoglobinemia sometimes may go unnoticed due to its relatively low occurrence, but cause fatal results [4]. However, it should be suspected in patients who have undergone outpatient procedures using topical local anesthetics, patients taking medications like dapsone, nitrates, and patients exposed to industrial agents. During preoperative interview, our patient told the history of unexpected cardiac arrest and the list of drugs to be avoided. Although we couldn't determine the exact agents used before, we suspected it to be methemoglobinemia due to prilocaine for local anesthesia via infiltration, and a continuous infusion of GTN for intentional hypotensive anesthesia. By this information, we preferred to use a ß-blocking agent, esmolol hydrochloride, for blood pressure control during surgery. As an alternative, in order to atenuate sympathetic adrenergic response during endotracheal intubation and due to surgical stress we`ve used esmolol hydrochloride, a short-acting and cardioselective β-blocker. We have chosen esmolol due to its advantage of ultra- short half-life (~9 min.) since it is metabolized by erythrocytes-esterases; and it has been shown to decrease the incidence of myocardial ischemia and arrhythmias in cardiac surgery [5,6].

As far as we reviewed, there isn't an adult case with methemoglobinemia undergoing cardiopulmonary bypass. As cardiac surgery requires a fine balance between oxygen demand and supply to the tissues, it is generally agreed that avoiding hypoxia and acidosis with adequate blood flow would be critical for perioperative care.

When methemoglobinemia is detected, offending agent should be discontinued immediately and supplemental oxygen should be started. The methylene blue given in a dose of 1-2 mg/kg intravenously (IV) over 5 minutes is standard treatment option. This dose can be repeated in 30-60 minutes if significant symptoms or high levels persist. Methylene blue works as an electron donor to reduce MetHb to Hb with the aid of NADPH-MetHb reductase enzyme (minor pathway) [7].

In the case of possible hypoxia or hemodynamic instability, methylene blue was ready in the operative room, but by avoiding the use of oxidizing agents we managed not to have MetHb related complications in our case.

Conclusion

A detailed preoperative patient evaluation, and close communication and collaboration among the surgeon, anesthesiologist and hematologist provide safe and optimum perioperative management. The anesthesiologist should avoid the use of oxidizing agents

and maintain O_2 carrying capacity of the arterial blood by ensuring high O_2 concentration in the inhaled gas in patients with suspected methemoglobinemia during general anesthesia.

Conflict of Interest

The authors declare no conflict of interest.

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