Diagnostic Dilemma: Low Oxygen Saturation during Cardiac Surgery

Abstract

We report a case of rheumatic heart disease with severe mitral stenosis having cyanosis and low oxygen saturation on pulse oximetry. The findings of clinical examination and low values on pulse oximetry were inconsistent with the findings of normal partial pressure of oxygen and oxygen saturation on arterial blood gas analysis, leading to diagnostic dilemma. In such clinical scenario, the anesthesiologist should be aware and vigilant about the differential diagnosis of low oxygen saturation on pulse oximetry.

Keywords: *Arterial blood gas, oxygen saturation, pulse oximetry*

**Suruchi Ladha,
Shivani Aggarwal,
Usha Kiran,
Arindam Choudhary,
Poonam Malhotra
Kapoor,
Ujjwal Kumar
Choudhary[1]**

*Departments of Cardiac
Anaesthesia and [1]Cardiothoracic
and Vascular Surgery, CNC,
AIIMS, New Delhi, India*

Introduction

One of the primary responsibilities of cardiac anesthesiologist is to ensure the adequate delivery of oxygen to the tissues of the body. In our clinical practice, when we encounter a patient with low oxygen saturation, cyanosis, and dyspnea, we usually focus on the cardiovascular system, respiratory system, and airway in search of the cause. However, sometimes, in patients who are undergoing cardiac surgery, the cause of low oxygen saturation on pulse oximetry may not be easily explainable or attributed to the cardiopulmonary cause, and the anesthesiologist should be vigilant about some rare differential diagnosis even in the presence of cardiac disease.[1] We report a case of severe mitral stenosis with diagnostic dilemma of low oxygen saturation on pulse oximetry.

Case Report

We report a case of a 28-year-old male diagnosed as a case of rheumatic heart disease with severe mitral stenosis and moderate tricuspid regurgitation scheduled for mitral valve replacement. Preoperatively, the patient was dyspneic at rest. The pulse oximetry showed saturation of 85%–90% with supplemental oxygen and reverse Trendelenburg position. We attributed this to severe pulmonary hypertension due to mitral stenosis although the chest was bilaterally clear on auscultation. Inside the operating room, routine monitoring

by electrocardiography, noninvasive blood pressure, and pulse oximetry was done. Pulse oximetry continued to show saturation of 85%–90% on supplemental oxygen. Under local anesthesia, peripheral venous and arterial lines were secured. The arterial blood sample was unusually dark red, and in view of low oxygen saturation values on pulse oximetry, the cause of dark red blood was attributed to the cardiopulmonary system. On the contrary, the arterial blood gas (ABG) analysis revealed uncompensated metabolic acidosis with pH 7.23, normal PO_2 of 270 mmHg, with 100% oxygen saturation, PCO_2 of 23 mmHg, HCO_3 of 10.8 meq/L, and base excess of 10.8 meq/L. This was paradoxical in a mitral stenosis patient who should have developed respiratory acidosis with low partial pressure of oxygen in such scenario. Symptomatic treatment was given with 100% oxygen and injection sodium bicarbonate. After induction of anesthesia, the diagnostic dilemma continued with the pulse oximetry still showing low oximetry readings on fractional inspired oxygen (FiO_2) of 1.0 and the subsequent ABG analysis (on GEM Premier 3000) showing pH of 7.25, PO_2 507 mmHg, SaO_2 100%, PCO_2 39 mmHg, HCO_3 17.1 meq/L, and base excess - 9.5 meq/L. There was a difference or gap between the saturation measured by pulse oximetry and ABG. We considered the possibility of persistent metabolic acidosis in low cardiac output state although the patient had no clinical signs of poor

Address for correspondence:
Dr. Shivani Aggarwal,
Department of Cardiac
Anaesthesia, CNC,
AIIMS, New Delhi, India.
E-mail: drshivani.del@gmail.com

perfusion, cold extremities, hypotension, or decreased urine output. Hemodynamic condition was stable throughout the perioperative period. After aortic cannulation, it was noted that the arterial blood was still unusually dark red to chocolate brown [Figure 1]. The FiO_2 of the oxygenator was 0.8 on the initiation of cardiopulmonary bypass (CPB). The ABG analysis revealed the same scenario, i.e., pH 7.3, PCO_2 42 mmHg, PO_2 243 mmHg, HCO_3 16.4 meq/L, base excess - 11.1 meq/L, and SaO_2 99.9%. This was confusing clinical scenario as with high FiO_2 and adequate flows on CPB; the metabolic acidosis should have been corrected. FiO_2 was then increased to 1.0 and repeat ABG was done on FiO_2 of 1.0. A mixed venous sample was also sent to assess the oxygen extraction. The venous sample had PO_2 of 44 mmHg and SvO_2 of 76%; thus, oxygen extraction was 24% which was also within normal range However, this time we used a different ABG machine (Cobas b 221) for analysis which also detects abnormal hemoglobins. The results were the same with pH 7.4, PCO_2 33 mmHg, PO_2 401 mmHg, HCO_3 20.9 meq/L, base excess - 3.2 meq/L, and SaO_2 100% but with methemoglobin (MHb) concentration of 28.1%!! The patient's MHb level was 0.28 proportion of the total hemoglobin (28.1%).

High FiO_2 of 0.8 was used during CPB as MHb interferes with oxygen binding. The patient was weaned off CPB successfully on inotropic support of dopamine (5 μgm/kg/min) and milrinone (0.5 μgm/kg/min). Post-CPB, the patient continued to have persistent metabolic acidosis with high MHb levels and normal oxygenation on ABG. Postoperatively, methylene blue (1 mg/kg of 1% solution [10 mg/mL]) was used which helped in decreasing the
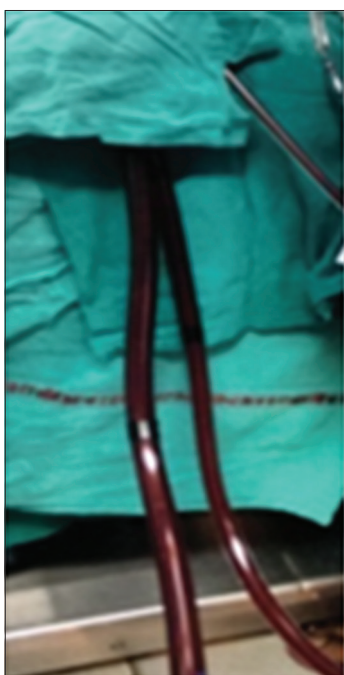

Figure 1: Dark red-colored blood in both arterial and venous line during cardiopulmonary bypass

MHb level. The patient had an uneventful postoperative recovery.

Discussion

The entire clinical scenario described above can be explained with the diagnosis of methemoglobinemia in a cardiac patient. The patient had cyanosis, low saturation on pulse oximetry, and metabolic acidosis because of methemoglobinemia which we were attributing to mitral stenosis. Patients with cyanosis due to cardiac disease who receive supplemental oxygen have a low partial pressure of oxygen and a low calculated oxygen saturation, but patients with methemoglobinemia have a high partial pressure of oxygen despite cyanosis and normal calculated oxygen saturation as seen in our patient.[2]

Methemoglobinemia (congenital or acquired) occurs when red blood cells contain MHb at levels higher than 1%. MHb results from the presence of iron in the ferric form instead of the usual ferrous form.[3] This results in a decreased availability of oxygen to the tissues. Excess MHb leads to cyanosis, impaired aerobic respiration, metabolic acidosis, and in severe cases, death.[3] The presence of persistent metabolic acidosis in our patient was due to methemoglobinemia.

The most common clinical measures of blood oxygen levels are the pulse oximetry-derived oxygen saturation and the ABG-derived PO_2 and SO_2. However, neither of these are adequate for detecting or measuring MHb. Conventional pulse oximetry can provide inaccurate readings in the presence of abnormal hemoglobins – MHb, sulfmethemoglobin, and carboxyhemoglobin.[4,5] Pulse oximetry measures the relative absorbance of two wavelengths of light (660 nm and 940 nm) that correspond to the absorption of oxyhemoglobin and deoxyhemoglobin, respectively. Ralston *et al.* reported that MHb has approximately the same absorption coefficient at both wavelengths, and if enough MHb is present, the absorbance ratio is approximately 1.0, which corresponds to an 85% saturation reading by pulse oximetry which was seen in our case.[4] It is reported that when MHb levels reach 30%–35%, the light absorbance reaches a plateau and the pulse oximeter reading becomes stable in the 82%–86% range.[4-6] ABG values can also be misleading.[7] The arterial PO_2 is a measure of dissolved oxygen and does not directly correlate with oxygen molecules bound to hemoglobin. This explains the normal PO_2 values found in our patient. The hemoglobin O_2 saturation level may also be unreliable as an ABG analyzer calculates this level from the pH and PCO_2 values, using the Henderson–Hasselbalch equation. This calculation assumes the presence of normal hemoglobin. The presence of MHb shifts the oxygen-hemoglobin dissociation curve to the left, resulting in false elevations of the O_2 saturation levels. One possible clue to the diagnosis of methemoglobinemia is the presence of a "saturation gap." This occurs when there

is a difference between the SO_2 that has been measured by means of pulse oximetry and the SO_2 that has been calculated by ABG analysis.[7] This saturation gap of >5% in our case helped us in suspecting methemoglobinemia in our patient and doing a detailed evaluation of different types of hemoglobin.

Coximeters are now being used for diagnosis of methemoglobinemia.[1] Co-oximetry, which measures light absorbance at four different wavelengths, using spectrophotometry, is one of the most accurate ways of measuring MHb levels. Since cooximeters are not routinely used and MHb levels are not routinely performed on ABG analyzers, in case of clinical suspicion, advanced ABG analysis machines should be used for timely detection of methemoglobinemia in the presence of associated cardiac disease[4] as we did in our case. The combined use of co-oximetry and blood gas analysis machines that measure oxygen saturation values will however be more accurate.

Once the diagnosis of methemoglobinemia was made, prompt treatment was initiated with methylene blue. Intravenous methylene blue is the first-line antidotal agent.[3] It accelerates the enzymatic reduction of MHb by nicotinamide adenine dinucleotide phosphate-MHb reductase and also reduces to leucomethylene blue that, in turn, reduces MHb. An infusion of dextrose may be used along with methylene blue as it upregulates the glycolytic cycle which is a major source of NADH in erythrocytes. Exchange transfusion, hemodialysis, and hyperbaric oxygen treatment are the second-line options. The treatment aims at restoring the oxygen-carrying capacity of blood and removal of oxidizing agent, if any. As cardiac anesthesiologist, we use several medications that have been associated with methemoglobinemia. The commonly used drugs such as nitroglycerine, sodium nitroprusside, nitrous oxide, metoclopramide, and local anesthetic (lignocaine, prilocaine, and benzocaine) should be avoided in such patients.[8-10] Our patient had no history of any such drug intake in the preoperative period. However, development of pulmonary hypertension required the use of vasodilators such as nitroglycerine in the postoperative period, but due to the presence of methemoglobinemia, drugs having nitrate/nitrite group were avoided and milrinone was preferred while weaning the patient from CPB.

This case highlights the importance of having a high index of suspicion of methemoglobinemia when there is clinical cyanosis with low pulse oximetry values not responding to conventional oxygen supplementation and high PO_2 values on ABG analysis. This is especially important in patients with cardiac disease who may already have low oxygen saturation. Methemoglobinemia, although rare, should be included in the differential diagnosis of cyanosis and low oxygen saturation on pulse oximetry. Proper perioperative care is only possible when a correct diagnosis is reached in situations causing a diagnostic dilemma between low oxygen saturation and clinical findings. The knowledge and awareness of the condition, appropriate monitoring, avoidance of the precipitating factors, and availability of antidote are key factors in managing a case of methemoglobinemia.

Conflicts of interest

There are no conflicts of interest.

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