ORIGINAL RESEARCH PAPER

Anaesthesiology

INCIDENTAL METHEMOGLOBINEMIA DETECTED IN A CASE OF SEVERE MITRAL STENOSIS WITH MODERATE PULMONARY HYPERTENSION POSTED FOR MITRAL VALVE REPLACEMENT. -A CASE REPORT

KEY WORDS:
methemoglobinemia, pulmonary hypertension, low oxygen saturation

Dr Kayalvizhi K B MD Assistant professor Tamilnadu Government Multi Super Speciality hospital, Chennai.

Dr Kalaivani Nadar* MD Assistant professor Tamilnadu Government Multi Super Speciality hospital, Chennai.*Corresponding Author

Dr L Parthasarathy MD Professor and HOD Tamilnadu Government Multi Super Speciality hospital, Chennai.

Da, Poovarasu MD PG resident Tamilnadu Government Multi Super Speciality hospital, Chennai.

ABSTRACT

Perioperative methemoglobinemia is overlooked as a cause for low oxygen saturation. Especially in a cardiac patient posted for valve replacement, the commonest cause for decreased oxygen saturation will be thought as pulmonary hypertension or pulmonary edema. Other factors like methemoglobinemia can also be a causative factor. Hence a high degree of suspicion is required to investigate uncommon causes of low oxygen saturation. A co oximeter is very valuable in these cases.

INTRODUCTION

Methemoglobinemia is a hemoglobinopathy caused by high levels of methemoglobin resulting from oxidation of ferrous to ferric state in hemoglobin. It is an uncommon but potentially fatal disorder. Methemoglobinemia causes interference with normal oxygen delivery. We present a case of incidental methemoglobinemia detected in a case of rheumatic heart disease with severe mitral stenosis and moderate pulmonary hypertension posted for mitral valve replacement.

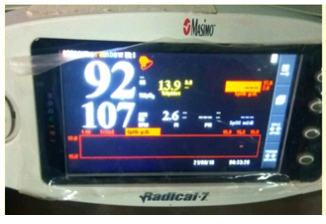
CASE HISTORY

A 45-year-old female a case of Rheumatic heart disease with severe mitral stenosis and moderate pulmonary hypertension was admitted in our hospital for elective mitral valve replacement surgery. She was a known schizophrenic patient on antipsychotic drugs (Olanzapine, Carbamazepine and Risperidone)

Her laboratory investigations including hemogram, coagulation profile, serum electrolytes, renal and liver function tests were normal. ECG showed atrial fibrillation, non-specific ST T changes, chest x-ray showed straightening of left heart border, Echocardiogram showed MVOA 1.0 cm2, Left Ventricular Internal Diameter (LVID) during diastole and systole 4.6/3.1 with Ejection fraction 63 %, Pulmonary Arterial pressure around 40 mm Hg.

After shifting to operation theatre, Standard monitors like ECG, NIBP, Pulse oximetry were applied. Room air saturation was 94%. Supplemental oxygen was given- 5L /min with venturi mask. Still saturation remained at 95%. Her vitals were stable with a Heart rate of 70/min, BP 110/70 mmHg and ECG-Atrial Fibrillation with controlled ventricular rate. Patient was on digoxin, frusemide, Aldactone and verapamil. On auscultation chest was clear and no added sounds. Under local anesthesia radial artery cannulation done, ABG sent for analysis. Arterial gas showed pH 7.36, high PaO2 of 245, pCO2 40, HCO3 22, with Fio2 0.6 . Preoxygenation and induction done using Inj fentanyl 3mcg/kg, thiopentone 5mg/kg, lidocaine 1.5mg/kg and atracurium 0.5mg/kg. Patient intubated with 7mm cuffed oral endotracheal tube. Bilateral air entry checked, was equal. Airway pressures were normal, and chest was clear. Etco2 was also normal with rectangular waveform. Still saturation was around 94 to 96% with 100% FIO2 at 7L/min. There was no desaturation during intubation. Since this was a case of mitral stenosis with pulmonary hypertension we thought pulmonary hypertension to be the cause for low saturation and started her on nitroglycerin infusion(10ug/kg/min). The saturation remained the same -around 94-95% .

After sternotomy we found that right atrium and right ventricle appeared normal and not dilated. Arterial blood gas again showed higher PaO2 and no acidosis. Hemodynamics were stable throughout and after ensuring adequate anticoagulation, cardiopulmonary bypass commenced, and mitral valve replacement done. Weaning from the cardiopulmonary bypass was also uneventful. The only consistent finding was little lower saturation of 94 % with normal or high paO2 on ABG.The intra-operative ABG after weaning from Cardiopulmonary bypass was pH 7.36, pCO2 30, pO2 220, hCO3 26 with FIO2 of 0.8 and Oxygen at 3L/min via flowmeter. Now we noticed that arterial blood was chocolate brown in color. There was no change in color of urine. We used a co oximeter (Masimo Radical 7) to evaluate.it showed elevated levels of met hemoglobin (13. 9%).However there was no cyanosis, systemic acidosis or hypotension since levels were not much elevated to produce a fatal picture. Use of nitroglycerin and lidocaine might have exacerbated the condition. None of the antipsychotics she used could exacerbate this condition. However, since arterial blood gases always had higher pao2 she did not require active intervention. After weaning from pump, we minimized on use of NTG and used esmolol instead. The diagnosis was further confirmed by spectrophotometry which showed elevated level of3.5% of methemoglobin.



This is a co-oximeter image showing methemoglobin level of 13.9 and saturation of 92% taken intraoperatively.

This is co-oximeter image taken on 3rd postoperative day shows methaemoglobin level of 9.3, obtained after eliminating the precipitating drugs.

DISCUSSION

Methemoglobinemia is formed when the Fe in hemoglobin is oxidized from ferrous to ferric state. Methemoglobinemia moves the ODC curve to the left[1]. Methemoglobinemia presenting in

cardiac surgery can be mistaken for pulmonary hypertension. Methemoglobin has minimum oxygen carrying capacity, hence in cases with higher levels of HbM above 30% there may be cyanosis, systemic acidosis and severe hypotension[2].Methemoglobin levels below 30% cause no symptoms. 30 to 50% cause symptoms and signs of oxygen deprivation like cyanosis, chest pain, organ ischemia, conduction abnormality, dysrhythmias. Above 50 % can result in coma and death[3].

The point to differentiate is that in pulmonary hypertension PaO2 is also low and requires frequent bagging with low cardiac output, if we use a Swan Ganz catheter we can easily pick a high pulmonary arterial pressure and they are corrected with infusions of nitroglycerine, dobutamine, or milrinone. In our case we had used only central venous pressure catheter and it was not raised. As a routine unless it is severe pulmonary hypertension we don't use Swan Ganz catheter. Persistence of high PaO2 levels and low saturation suggests the diagnosis of methemoglobinemia. Use of a co oximeter greatly aids in diagnosis. In our case we further confirmed the diagnosis later by spectrophotometry.

In our case, it was acquired methemoglobinemia as the presentation was in adulthood after exposure to oxidizing agents. Exposure to certain drugs or chemicals cause faster oxidation of hemoglobin to methemoglobin. Commonly implicated oxidative substances are dapsone, lidocaine, nitroglycerine, sulfonamides, phenytoin [4,5]. In our case we had used lidocaine and nitroglycerine which might have exacerbated the condition. The other factors which also exaggerate methemoglobinemia are metabolic acidosis, anemia and even cardiopulmonary bypass itself Management involves removal of the offending agent, supplemental oxygen. In patients with methemoglobin levels >30%, treatment is iv methylene blue 1 to 2 mg/kg i.v over 5 mins[6]. Total dose should not exceed 7 - 8 mg/ kg. Our patient was on anti-psychotic medication. Though they do not exacerbate the condition, we found that when methylene blue is administered to patients on serotonergic medications, there is chance of CNS toxicity known as serotonin syndrome[7]. Thankfully we did not require use of methylene blue. Ascorbic acid also reduces methemoglobin directly. Our primary goal is to avoid tissue hypoxia. Acidosis should be corrected. ECG must be closely monitored for any signs of ischemia. Patients with HbM are very sensitive to oxidizing agents hence should be avoided. Methylene blue acts through the methemoglobin reductase pathway and requires action of G6PD.

CONCLUSION:

Methemoglobinemia should be considered as differential diagnosis whenever there is low oxygen saturation with high PaO2, not responding to supplemental oxygen. Use of co oximeter not only detects but also quantifies methemoglobin levels. Avoidance of precipitating factors, appropriate monitoring and use of antidote if required, help in managing a case of Methemoglobinemia.

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