Methemoglobinemia caused by accidental poisoning by nitric oxide - case report

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ABSTRACT

The aim of this paper is to present a case of methemoglobinemia induced by accidental poisoning by nitric oxide, in a male infant at the age of 3 months and 4 days. Methemoglobinemia is a disease in which the hemoglobin is in the oxidized form and can not bind oxygen, and becomes manifested when erythrocytes contain more than 1% of methemoglobin. This condition can be idiopathic or acquired, and requires sub-specialist diagnostic evaluation and treatment.

Keywords: methemoglobinemia, nitric oxide, infant.

INTRODUCTION

Methemoglobinemia is a condition characterized by increased quantities of hemoglobin in which the iron of heme is oxidized to the ferric (Fe3+) form, thus making it unable to transport oxygen. (1, 2) This condition can be a result of a genetic deficiency in erythrocyte metabolism or structural changes in hemoglobin, or may be of acquired character (exposure to toxic or pharmacological agents). (2)

CASE REPORT

A child (aged 3 months and 4 days) was sent from the Cantonal Hospital Travnik under diagnosis of bronchopneumonia and cardiorespiratory insufficiency. The illness started on the day before the admission with severe breathing, and after hospitalization it was advised to continue treatment in the Pediatric Clinic, Clinical Center University of Sarajevo due development of the global respiratory insufficiency. At admission, the child was disturbed, tachypneic, “catches air”, occupies a forced position – opisthotonus. The skin has been changed by the type of dermatitis with excoriations. Ears were protruding, with delayed reaction to light, nose foramen had a brown color. Methemoglobinemia was suspected, laboratory findings were performed and the value of FmetHb (fraction of MetHb in Hb) was 22.3%. Since there was no possibility to use methylene blue as therapy, exsanguination transfusion was indicated. The procedure started at 20:05 hours, and was completed at 22:45 hours (without complications and the patient was cardiovascularly stable during the procedure) (Table 1). During sampling it was noticed that blood had a brown color that was characteristic of methemoglobinemia, and that was not the case with blood samples taken before receiving nitric oxide (NO) in therapy. That made a suspicion that accidental poisoning with NO occurred. After completion of exsanguination transfusion, generalized cyanosis disappeared, Fmet Hb values were no longer increased and the patient had no clinical symptoms (which is inherited metabolic disturbance of the enzyme or the disorder of structure of hemoglobin). On the third day of hospitalization, the

Table 1. Laboratory findings before and after exsanguination transfusion

<table>
<thead>
<tr>
<th></th>
<th>At the beginning (20:05)</th>
<th>After completion of exsanguination transfusion (22:45)</th>
</tr>
</thead>
<tbody>
<tr>
<td>RBC (x1012/L)</td>
<td>3.23</td>
<td>4.72</td>
</tr>
<tr>
<td>Hb (g/L)</td>
<td>78</td>
<td>128</td>
</tr>
<tr>
<td>Hct (L/L)</td>
<td>0.39</td>
<td>0.41</td>
</tr>
<tr>
<td>WBC (x109/L)</td>
<td>8.1</td>
<td>10</td>
</tr>
<tr>
<td>Plt (x109/L)</td>
<td>210</td>
<td>138</td>
</tr>
<tr>
<td>FmetHb (%)</td>
<td>22.3</td>
<td>2.1</td>
</tr>
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</table>
general condition was more stable, poractant alfa was repeated, and ventilatory support was reduced. Oral intake of the food was, the sedation was excluded and the child was successfully extubated on the seventh day, breathing for two days with the aid of O2 then without oxygen and was discharged in good general condition.

DISCUSSION

NO is indicated in combination with ventilation support for treatment in the following cases: in infants ≥ 34 weeks of gestational age with hypoxic respiratory insufficiency associated with clinically or echocardiographically demonstrated pulmonary hypertension to improve oxygenation and reduce the need for extracorporeal membrane oxygenation and also as part of the treatment of perioperative and postoperative pulmonary hypertension associated with heart surgery. (3, 4) Despite rapid inactivation by circulating hemoglobin, inhaled nitric oxide exerts effects outside the lung, including blocking platelet aggregation, causing methemoglobinemia. (3) Overdose with NO is manifested with an increase of methemoglobin and NO2. Increase of methemoglobinemia reduce blood oxygen supply capacity. Methemoglobinemia that cannot be resolved after reduction or discontinuation of therapy can be treated, depending on clinical evaluation, by intravenous administration of vitamin C or methylene blue or blood transfusions. (1, 5)

CONCLUSION

Although used as a pharmacologic agent, nitric oxide may also have a toxic effect, which must be urgently treated. For therapeutic purposes, it is necessary to measure the level of methemoglobin within one hour after the onset of therapy. Severe, acquired methemoglobinemia resulting from exposure to drugs and poisons has to be treated by intravenous administration of large amounts of Methylene blue (2 mg/kg - repeat if necessary). Such treatment reduces the amount of methemoglobin by 50% within one hour after application. If for some reason it is not possible to apply this therapy it is necessary to perform exsanguination transfusion.

REFERENCES