Methemoglobinemia in children: how to explain the results?

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All pigments of hemoglobin (oxyhemoglobin, methemoglobin, sulphemoglobin and carboxyhemoglobin) are of clinical importance and each has a laboratorial characteristic in the absorption spectrum. Methemoglobin, in which the ferrous iron atom of the heme group has been oxidized to a ferric iron atom, is unable to carry molecular oxygen. Various red cell enzymes convert methemoglobin back to oxyhemoglobin and keep the level of this pigment between 1% and 3%. High levels of methemoglobin (> 5%) are associated with chocolate-colored blood, cyanosis, headaches, tachycardia, dyspnea and tachypnea and in severe methemoglobinemia coma and death can occur.[1] Newborns are more susceptible to methemoglobinemia because fetal hemoglobin (Hb F) is easily oxidized.[2] Methemoglobin may arise from three different causes:

1. An excessive formation of methemoglobin due to contamination by chemical products, such as phenacetin, nitro or amino derivatives of benzene, derivatives of sulfur, etc. These products cause toxic methemoglobinemia.[3]

2. Inherited enzyme deficiencies including superoxide dismutase (SOD), glutathione peroxidase (GPx), catalase and NADH-linked methemoglobin reductase or diaphorase. These enzyme deficiencies are inherited as Mendelian recessive conditions and are responsible for dramatic cases of cyanosis and hemolytic anemias.[4]

3. Methemoglobinemia due to abnormalities of the beta and alpha globins, especially those near to the heme group region. This abnormal form of oxidized hemoglobin is referred as hemoglobin M (Hb M) and there are at least six different types: Hb M Boston, a variant hemoglobin caused by a mutation in alpha globin (alpha 58 His → Tyr), Hb M Iwate (alpha 87 His → Tyr), Hb M Saskatoon (Beta 63 His → Tyr), Hb M Zurich (Beta 63 His → Arg), Hb M Hyde Park (Beta 92 His → Tyr) and Hb M Milwaukee (Beta 67 Val → Glu).[1,5]

The article "Reference values for methemoglobin concentrations in children"[6] published in this issue of the *Revista Brasileira de Hematologia e Hemoterapia* showed that the methemoglobin levels in 6- to 10-year-old children are higher than those of healthy adults. The authors suppose that the difference in levels is due by a smaller amount of soluble cofactor cytochrome b5 and less activity of cytochrome b5 reductase in the red blood cells of children. However these enzymatic activities were not evaluated by the authors. So, should not factors (school, food, water, etc) in the environmental where the children of this investigation live be considered as a possible cause of the increased levels of methemoglobin?[3,4,7]

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