

International Perspective from Singapore on “Methemoglobinemia and Sulfhemoglobinemia in Two Pediatric Patients after Ingestion of Hydroxylamine Sulfate”

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Accidental ingestional poisoning among pediatric patients is a prevalent problem. In the absence of a well-designed national injury and poisoning surveillance system, cases often go unreported. Underreporting also occurs because many pediatric ingestions are trivial and not referred to a hospital for further medical management.¹ To reduce health risks posed by common household products, emphasis and enforcement of regulations specifying child-safe packaging are required.²

The authors described two cases of methemoglobinemia (metHbemia) and sulfhemoglobinemia (sulfHbemia) following ingestion of hydroxylamine sulfate, which were treated with methylene blue and exchange transfusion. Drugs or chemicals that cause sulfHbemia can also cause metHbemia, although it is not understood why the same chemical causes metHbemia in one person and sulfHbemia in another.³ Other ingestants that may cause methHbemia and sulfHbemia are dapsona, metoclopramide, phenacetin and phenazopyridine.

Emergency physicians (EP) in some developing countries may not have access to lab equipment with advanced co-oximeter capabilities to differentiate the two types of dyshemoglobinemia. In a time-pressured situation, some bedside investigation methods may be useful in guiding decisions for specific therapy like methylene blue in a cyanosed patient with suspected poisoning. One method, the filter paper test, helps to distinguish deoxyhemoglobin from dyshemoglobin as the darkly colored blood changes to bright red after blowing some oxygen over it. No changes occur with metHb or sulfHb. To distinguish metHb from sulfHb, the addition of a few drops of potassium cyanide changes the chocolate brown of metHb to bright red as cyanometHb is formed. No reaction occurs with sulfHb.⁴ Therapeutic response to methylene blue will also aid in the diagnosis of underlying metHbemia. The response is usually fairly rapid, within 30 minutes to one hour.

EPs should consider several differentials for apparent metHbemia that does not respond to methylene blue treatment.

These include older equipment incapable of distinguishing sulfHb from metHb due to limited co-oximeter capability; Hb M disease prone to metHb formation that resists reduction; glucose-6-phosphate dehydrogenase (G6PD) deficiency; NADPH metHb reductase deficiency; poisoning with oxidizing compounds that have enterohepatic circulation, which causes prolonged elevation of metHb; and overdosing of methylene blue itself, which is an oxidizing agent.^{3,4}

Finally, G6PD deficiency, one of the most prevalent disease-causing mutations worldwide, has several variants in Asia. Patient's status will influence decision when using methylene blue as treatment for metHbemia because methylene blue itself may induce hemolysis (through development of Heinz bodies) and cause paradoxical metHbemia, especially in G6PD deficient patients.⁴

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