Case Report

Methylene blue-A double-edged sword in the treatment of methemoglobinemia in a patient with emamectin benzoate poisoning: A case report

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ABSTRACT

Emamectin benzoate (EB), a widely used avermectin-derived pesticide, rarely causes mammalian toxicity but can induce severe methemoglobinemia following acute exposure. We report a case detailing the management of EB-induced methemoglobinemia, complicated by methylene blue-induced hemolytic anemia in a non-G6PD-deficient patient. A 34-year-old male presented with altered sensorium, hypoxemia, and severe methemoglobinemia (63%) 6 h post-ingestion of 15 mL EB (1.9%). While early intubation and targeted pharmacotherapy (methylene blue and Vitamin C) successfully normalized his methemoglobin levels, his clinical course was complicated on day six by non-G6PD-deficient hemolytic anemia (hemoglobin drop: 13.8–6.5 g/dL). The patient recovered fully following supportive therapy and red blood cell transfusion. This case demonstrates that while methylene blue effectively reverses EB-induced methemoglobinemia, it can precipitate severe, delayed hemolytic anemia even in G6PD-normal patients. This suggests a potential synergistic hematologic toxicity between EB and methylene blue. Clinicians should maintain prolonged vigilance for hemolysis and carefully weigh alternative therapies for methemoglobinemia.

Key words: Emamectin benzoate, Hemolytic anemia, Methemoglobinemia, Methylene blue

Emamectin Benzoate (EB) is a semi-synthetic pesticide from the avermectin family, specifically designed to target lepidopteran insects [1]. EB exhibits a strong affinity for gamma-aminobutyric acid receptors. Stimulation increases the influx of chloride ions into neuronal cells, resulting in cellular damage and impaired nerve impulse transmission [2]. Clinical manifestations include nausea, vomiting, and drowsiness [3].

This case highlights methemoglobinemia as a presentation of EB poisoning and the risk of hemolysis with methylene blue even in patients with normal G6PD levels in EB poisoning.

CASE REPORT

A 34-year-old gentleman came to the emergency room with an alleged history of consumption of 15 mL of EB (Srikar Sixer – contains 1.9% EB) (Fig. 1). He came to the emergency department 6 h after the consumption, after being taken to a local hospital. He had altered mental status and two episodes of non-bilious vomiting on arrival. On examination, he was drowsy with Glasgow Coma Scale (GCS) of E1V1M1, with tachycardia (pulse rate – 120 bpm), respiratory rate of 22/min, blood pressure of 100/70 mmHg, SpO_2 of 81% in room air, which did not improve with oxygen via a non-rebreathing oxygen mask. Other causes of hypoxia were ruled out. He was electively intubated due to low GCS and hypoxemia. He was shifted to the Intensive Care Unit (ICU) for further management.

Initial arterial blood gas (ABG) analysis showed a methemoglobin (MetHb) level of 63%. Initial blood tests revealed leukocytosis (18300/cumm), mildly elevated total bilirubin (1.9 mg/dL), hypophosphatemia, hypomagnesemia, and hypocalcemia. Chest X-ray and ECG were normal.

He received 60 mg of methylene blue infusion (1 mg/kg in 5% dextrose over 30 min). Serial ABGs were done to monitor MetHb levels. Urine color was found to be dark green after the first dose of methylene blue. After 10 h, his MetHb levels came down to 2%. His sensorium improved gradually, and he was extubated on day 2 of admission. Given persistent methemoglobinemia (13.8 % on day 2), he received a high dose of Vitamin

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C (1 g thrice daily), and methylene blue was repeated at 1 mg/kg.

On day 6 of admission, he was found to have hematuria with a significant drop in hemoglobin level from 13.8% to 6.5% (Fig. 2). G6PD level was normal. Hemolytic workup was done, which showed elevated lactate dehydrogenase (LDH), reticulocyte counts, and unconjugated hyperbilirubinemia, with peripheral smear showing a normocytic normochromic picture with neutrophilic leukocytosis. He was transfused with 2 units of packed red blood cells; following this, hemoglobin levels remained stable. As he improved clinically, he was shifted to the ward for the continuation of care. Psychiatry consultation was sought from the ward. He recovered completely, and he was discharged home after 12 days of hospital admission.

Table 1 shows the serial ABG and MetHb levels.



Figure 1: The compound Srikar Sixer containing emamectin benzoate at a concentration of 1.9% w/v



Figure 2: Hematuria on day 6 of admission

DISCUSSION

EB is considered harmless to humans (mammals) due to its limited ability to traverse the blood-brain barrier, the lack of glutamate-gated chloride channels in mammals, and its low affinity for mammalian chloride channels [4]. However, significant exposure to EB can result in life-threatening poisoning requiring intensive care admissions. Common clinical presentations include sore throat, gastrointestinal symptoms, central nervous system depression, aspiration pneumonia, and respiratory failure [5]. There are several case reports of EB poisoning causing clinically significant methemoglobinemia needing intervention [6,7]. There is no specific antidote available; management remains supportive care.

Methemoglobinemia is a potentially life-threatening illness characterized by a reduction in the oxygen-carrying capacity of circulating hemoglobin, resulting from the conversion of some or all of the four iron ions from the reduced ferrous [Fe^{2+}] state to the oxidized ferric [Fe^{3+}] state. This diminishes oxygen delivery to the tissues due to the leftward shift of the oxygen-hemoglobin dissociation curve, causing tissue hypoxia [8]. Methemoglobinemia can be congenital and acquired. Congenital methemoglobinemia is rare, usually caused by cytochrome b5 reductase deficiency. Acquired methemoglobinemia is more frequently encountered. The majority of instances result from unintentional exposure to a chemical or the application of topical or local anesthetics [8]. The management of methemoglobinemia involves eliminating the causative agent and administering the antidote, methylene blue (tetramethylthionine chloride).

Previous literature highlights only two cases of methemoglobinemia following EB exposure, neither of which resulted in mortality [6,7]. The standard management in these reports involved Vitamin C and methylene blue, with one case utilizing hemoperfusion to address indoxacarb co-poisoning. In line with these documented strategies, we successfully managed our patient's presentation using methylene blue and high-dose Vitamin C.

During the ICU stay, we noticed hematuria with dropping hemoglobin levels and elevated serum LDH, which were suggestive of hemolytic anemia, although G6PD levels were normal. Methylene blue is known to cause hemolytic anemia in patients with G6PD deficiency. In our patient, G6PD levels were normal, suggesting a possible role of both EB and methylene blue in

Table 1: The serial ABG and methemoglobin levels

Parameters	Admission	At 10 h	At 30 h	Day 2	Day 3	Day 4	Day 9
pH	7.407	7.419	7.601	7.437	7.467	7.440	7.530
Pco2	34.5	31.6	21.7	34.4	35.7	35.1	24.9
Po2	31.5	57.5	149	154	147	146	95.5
MetHb	63.1	2.2	13.8	1.5	8.2	5.9	2.7

MetHb: Methemoglobin, ABG: Arterial blood gas

precipitating hemolytic anemia. There is one case report in the literature that showed hemolytic anemia in patients with organophosphorus and nitrofurantoin poisoning after using methylene blue for 8 days in a patient with normal G6PD levels [9]. Kaidama and Aqlan showed that administering EB to pigeons significantly reduced hematological parameters, including hemoglobin, white blood cells, and platelets. The possible reason can be immunosuppression and the destructive effect of EB on red blood cells [10]. Our case is possibly the first in the literature demonstrating hemolytic effects of the EB compound.

CONCLUSION

EB poisoning is associated with a significant risk of methemoglobinemia. Treatment with methylene blue should be considered carefully in patients with EB poisoning, as there is a risk of precipitating hemolysis. Alternative treatment for methemoglobinemia should be considered in cases of significant hemolysis with methylene blue.

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