WebLog Journal of Toxicology

Case Report
Published: 13 Feb, 2026

Management of Methemoglobinemia due to Terpene-lactoid Poisoning: A Case Report

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Abstract

In today's world, herbal or "natural" insecticides are generally considered benign, but their toxic potential remains inadequately recognized. We report the case of a 75-year-old man who voluntarily took a commercially available herbicide containing terpenes, alkaloids, lactones and emulsifiers. He presented himself to the emergency department with repeated vomiting, altered sensors, progressive cyanosis and hypoxia, which did not improve despite additional oxygen. Initial clinical results differed from pulse oximetry measurements, prompting further investigation. Co-oximetric analysis of arterial blood gas revealed severe methemoglobinemia with a level of 56.6% of methaemoglobin, accompanied by significant lactic acidism (lactate 13.1 mmol/L). The patient underwent immediate gastric decontamination, airway protection and intensive support care. After confirmation of normal glucose-6-phosphate dehydrogenase levels, intravenous methyl blue was administered along with ascorbic acid and thiamine. After antidotal therapy, levels of methaemoglobin normalized rapidly, lactate concentrations declined, and the neurological state improved considerably. The patient was successfully liberated from ventilator support and recovered without residual sequelae. This case highlights the often-overlooked toxicity of herbal insecticides and emphasizes the importance of considering methemoglobinemia in patients with unexplained cyanosis and saturation gap, where early recognition and targeted treatment can save lives.

Keywords: Methemoglobinemia; Herbal Insecticide Poisoning; Terpenes; Methylene Blue; Lactic Acidosis

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Received Date: *13 Jan 2026*
Accepted Date: *11 Feb 2026*
Published Date: *13 Feb 2026*

Citation:
Joshi AS, Chavan S, Momin OMU, Sayyed AS. Management of Methemoglobinemia due to Terpene-lactoid Poisoning: A Case Report. WebLog J Toxicol. wjt.2026. b1306. https://doi.org/10.5281/ zenodo.18795798

Introduction

Herbal or "natural" insecticides are increasingly used to control domestic pests and are often considered an inherent safe alternative to synthetic pesticides [1]. These formulations usually contain mixtures of bioactive plant compounds, including terpenes, alkaloids, lactones and emulsifiers [2], which are intended to improve insecticide efficacy and product stability. Although widely available and good-to-market, evidence suggests that these constituents can have significant toxicity in humans after consumption or extensive exposure [3]. In particular, intentional or accidental ingestion of such products has been linked to various adverse effects, including severe methemoglobinemia, a life-threatening condition characterized by impaired oxygen transport [4].

Terpenes, particularly monoterpenes and sesquiterpenes, are highly lipophilic compounds that are rapidly absorbed from the gastrointestinal tract and systemically distributed [5]. They undergo hepatic phase I metabolism predominantly via cytochrome P450 enzymes, generating reactive electrophilic metabolites [6]. These intermediates can deplete intracellular glutathione, promote oxidative stress, mitochondrial dysfunction, and lipid peroxidation, ultimately resulting in hepatocellular damage [7]. Several terpenes can also cross the blood–brain barrier, producing neurotoxic manifestations such as agitation, seizures, confusion, and ataxia [8]. In addition, oxidative metabolites of certain terpenes have been implicated in red blood cell injury, creating a biochemical milieu conducive to the formation of methaemoglobin [3].

Alkaloids, a structurally diverse group of nitrogen-containing phytochemicals, are well recognized for their potent biological activities. Many alkaloids exert toxicity by interfering with cellular respiration, ion channels, and oxidative pathways [9]. Some plant-derived alkaloids and related compounds have been associated with oxidative stress on haemoglobin, leading to methemoglobinemia, haemolysis, and tissue hypoxia [4]. Furthermore, alkaloid-induced mitochondrial impairment may shift cellular metabolism toward anaerobic glycolysis, thereby contributing to lactic acidosis in severe cases of poisoning [9].

Lactones, including sesquiterpene lactones and other cyclic esters, are common constituents of herbal extracts used in insecticidal preparations. These compounds readily react with cellular nucleophiles and are associated with hepatotoxicity, cytotoxicity, and inflammatory responses [10, 12]. Experimental data suggest that lactones can exacerbate oxidative stress and impair mitochondrial function [11], mechanisms that may further potentiate hypoxic injury and metabolic acidosis [12].

Although often considered pharmacologically inert, emulsifiers and surfactants used to solubilize hydrophobic plant extracts can enhance the gastrointestinal absorption of toxic constituents and may independently cause mucosal irritation, vomiting, and systemic toxicity at high doses. By increasing the bioavailability of active compounds, these agents may amplify the overall toxic effects in mixed ingestions [13]. Collectively, the convergence of oxidative haemoglobin injury, mitochondrial dysfunction, and impaired cellular respiration provides a plausible pathophysiological basis for the development of methemoglobinemia and lactic acidosis following the ingestion of complex herbal insecticide formulations [3]. However, clinical descriptions of such toxicities remain scarce, and most poison surveillance systems do not routinely characterize the specific phytochemical constituents involved [4].

We report a case of a 75-year-old male who intentionally ingested a commercially available herbal insecticide containing terpenes, alkaloids, lactones, and emulsifiers, and subsequently developed methemoglobinemia with mild lactic acidosis and neurological symptoms. The patient responded to gastric decontamination and antidote therapy with methylene blue, supported by ascorbic acid and thiamine. This case highlights the underestimated toxic potential of natural insecticides and underscores the need for heightened clinical awareness and regulatory scrutiny, particularly in vulnerable elderly populations.

Case Presentation

A 75-year-old man was brought to ER at 5 pm by his family members with the chief complaint of ingestion of an organic bed bug killer (terpene alkaloid, lactoid) approximately an hour ago. He experienced nausea and vomiting after consumption until he reached the hospital. The patient had a staring look and stiffness of the extremities with unresponsiveness, as described by the relatives. In the ER, his physical examination showed PR 120/min, Spo_2 85% on RA and 100% with O_2 by mask on 10 L but still showing cyanosed peripheries BP was normal that is 140/70 and RR 14/min. In ABG it was found to be pO_2 was decreased i.e. 22.5 mmHg (normal- 80 to 100 mm hg), Fmeth was increased to 56.6% (normal: 0-2%) which gives the confirmed diagnosis as methemoglobinemia, in addition with lactic acidosis as cLac was increased to 13.1 (normal: 0.4- 0.8 mm/L) his pupil was approx. 2 mm and sluggishly reacting to light. He was afebrile, with normal cardiac sounds, bilateral respiratory sounds, and a soft, non-tender abdomen.

In the ER, the patient was administered Inj. Pantoprazole 40mg IV, Inj. Ondansetron 4 mg IV in NS 500 ml over 5 h. initially. The patient underwent poison management, including intubation for airway protection and gastric lavage for stomach wash with activated charcoal. The patient was shifted to the ICU, the vitals were HR was 100/min (ECG: sinus tachycardia), bp was 112/66 mmHg, CVS was normal, CT report suggested chronic white matter ischemic changes may be due to age-related cerebral atrophy, and pupil was now equally reacting to light, thus he was shifted to ICU for further management.

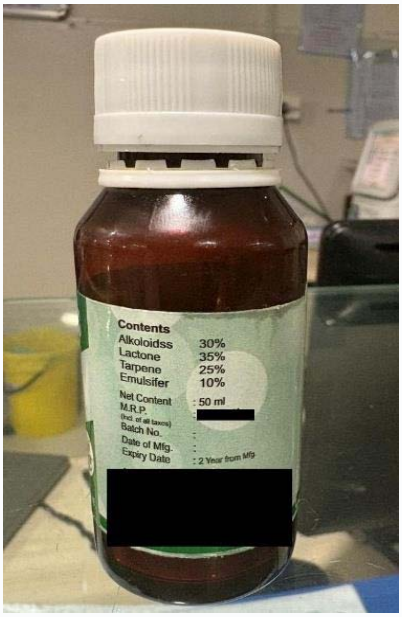

Figure 1: Content of poison.

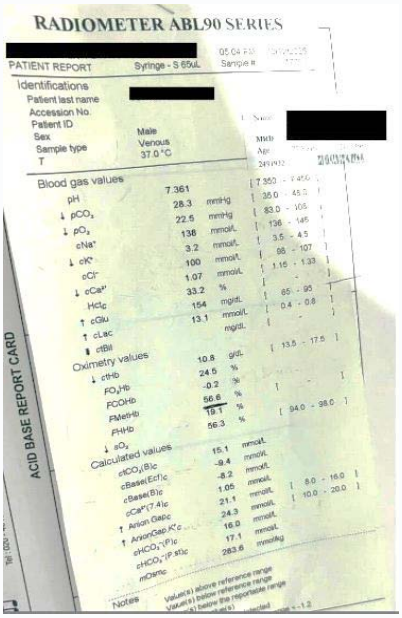

Figure 1: ABG in ER.

In the ICU, certain investigations were performed, such as chest X-ray and POCUS, both of which were normal. The BSL was 183 mg/dl, and the serum lactate level was 5.7 mm/L (normal range, 0.5–2.0 mm/L) to check oxygen delivery to the tissues. The patient was administered Inj. vit C 1 gm IV stat dose over 1 min, Inj. methylene blue (100 mg in 100 ml NS IV) over 10–15 min and Inj. thiamine 200mg IV BD. His glucose 6 phosphate dehydrogenase level was 30.1 U/gHb (normal range: 6.4-20.0 U/gHb) at 37°C which indicated that methylene blue was safe to administer. An additional ABG was performed after the administration of methylene blue, which showed that FmetHb decreased to 1.2% and cLac decreased to 2.6 mm/L. The pO_2 was 212 mmHg, which was slightly lower than that in the

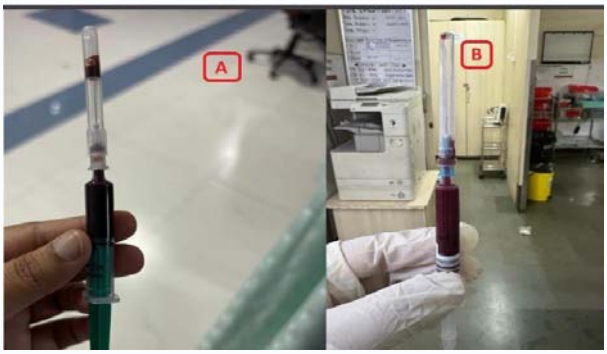

Figure 3: (A) Blood sample colour before Methylene blue. (B) Blood sample colour after Methylene blue.

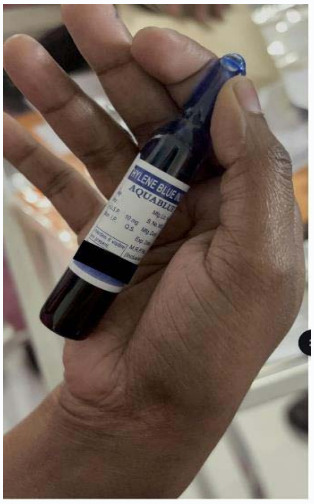

Figure 4: Vial of Methylene blue.

- Blood Gases ABG*	
pH	7.41 -
pC02	37 mm Hg
p02	212 mm Hg
Na+	131 mmol/L
K+	3.0 mmol/L
Cl	102 mmol/L
Ca ++	1.00 mmol/L
Hct	33 %
Glu	207 mg/dl
Lac	2.6 mmol/L
tBili	3.0 mg /dl
tHb	9.5 g/dl
O2Hb	96.5 %
COHb	0.5 %
MetHb	1.2 %
HHb	1.8 %
sO2	98.2 %
BEecf.	-1.1 mmol/L
tHb(c).	11.2 g/dl
BE(B).	-0.9 mmol/L
Ca++ (7.4).	1.00 mmol/L
AG.	9 mmol/L
sO2(c)..	99.8 %
HCO3(c).	23.5 mmol/L
HC03std..	24.2 mmol/L
Ventilator Settings	5+12 50%

Figure 5: ABG after administration of methylene blue.

previous sample; therefore, the fiO_2 was tapered in the ventilator to avoid oxygen toxicity. The patient was awake, conscious, oriented, obeying commands, and symptomatically better after the procedure.

Discussion

This case demonstrates a classical but rarely reported pattern of poisoning caused by a commercially available herbal insecticide, in which a mixed phytochemical formulation produced a predictable sequence of physiological failure—hypoxia, neurological dysfunction, and metabolic acidosis—through a single unifying mechanism: failure of oxygen delivery at the haemoglobin and mitochondrial levels.

The patient's initial presentation was dominated by cyanosis, altered sensorium, and low oxygen saturation that did not improve with high flow oxygen. This discordance between the administered oxygen and clinical hypoxia is the hallmark of methemoglobinemia rather than primary pulmonary pathology [14]. The markedly elevated methaemoglobin fraction (56.6%) explains why oxygen therapy alone was ineffective: although oxygen was present in the blood, it could not be delivered to tissues because haemoglobin was rendered functionally inactive [15]. This led to widespread cellular hypoxia, which was biochemically reflected by the severe lactic acidosis observed on admission. Neurological manifestations, such as staring, rigidity, and unresponsiveness, can be attributed to cerebral hypoxia rather than a primary neurotoxic insult. The rapid normalization of mental status following the reduction in methaemoglobin levels strongly supports this interpretation. If the symptoms were caused by structural brain injury or direct neurotoxicity, such a dramatic reversal would not have occurred within minutes of antidotal therapy [16]. Similarly, lactic acidosis was not due to shock, sepsis, or hypoperfusion, as the patient was hemodynamically stable with normal blood pressure and no signs of infection or circulatory collapse. Instead, it reflects cellular hypoxia caused by the failure of haemoglobin to release oxygen to tissues. This explains why lactate levels decreased rapidly once functional haemoglobin was restored, even before complete clinical recovery [17].

The response to methylene blue provides both diagnostic and therapeutic confirmation of this mechanism. Methylene blue bypasses impaired endogenous reduction pathways and directly restores the oxygen-carrying capacity of haemoglobin [18]. In this patient, methaemoglobin levels fell from 56.6% to 1.2%, and lactate levels decreased from critical to near-normal levels within a short period. This parallel improvement demonstrates that methemoglobinemia was the driver of both hypoxia and metabolic derangement, not a secondary phenomenon. The use of ascorbic acid and thiamine further supported recovery by facilitating redox balance and mitochondrial metabolism [3]. Their inclusion was particularly relevant given the profound metabolic stress indicated by the initial lactate level. Importantly, confirmation of adequate G6PD activity allowed for the safe administration of methylene blue, preventing treatment-induced haemolysis, which could have worsened hypoxia [19].

What makes this case clinically important is that the poisoning originated from a product marketed as "organic" and "safe." Unlike conventional pesticides, herbal insecticides are not widely recognized as causes of methemoglobinemia, which can delay diagnosis. Therefore, this case bridges an important gap between toxicology and clinical practice, showing that herbal insecticides can produce the same life-threatening hematologic toxicity as synthetic oxidizing agents and should be approached with the same urgency. Early co-

oximetry, prompt antidotal therapy, and aggressive supportive care are essential for patient survival.

In summary, this patient's rapid deterioration, dramatic biochemical abnormalities, and equally rapid recovery after methylene blue administration demonstrate a clear cause-effect relationship between herbal insecticide ingestion and severe methemoglobinemia with secondary lactic acidosis. This case underscores the need for heightened clinical suspicion, better labelling and regulation of herbal insecticides, and public awareness that "natural" does not mean non-toxic, particularly in vulnerable elderly populations.

Acknowledgement

The authors acknowledge the contributions of those directly involved in this work. **Dr. Ashwini Joshi** (Consultant, Medicine Dept., DMHRC) conceptualized the study, collected and analysed the clinical data, drafted the manuscript and approved the final manuscript. **Dr. Supriya Chavan** (Resident, Medicine Dept., DMHRC) contributed to patient evaluation, interpretation of clinical findings, and critical revision of the manuscript. **Osama M. U. Momin** (Intern, Pharmacy Dept, DMHRC) assisted with literature review, data validation, and manuscript editing. **Ajmat S. Sayyed** (Intern, Pharmacy Dept, DMHRC) provided supervisory input in literature review, data collection & validation and manuscript editing. All authors have reviewed and approved the final version of the manuscript and agree to be accountable for all aspects of the work.

Financial Support: This case report did not receive any financial support or funding from public, commercial, or not-for-profit agencies.

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