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Case report : pyridaben poisoning induced acute respiratory and circulating failure

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Abstract

Background Pyridaben, a commonly employed pyridazinone insecticide, is extensively utilized in safeguarding crops against insects and mites. Pyridaben is known to impede mitochondrial complex I activity, inducing mitochondrial impairment, which subsequently culminates in cellular hypoxia, cessation of glycolysis, swift lactic acid buildup, and the onset of lactic acidosis. Prolonged exposure to pyridaben has been demonstrated to elicit deleterious effects on neuronal cells. Furthermore, pyridaben has been associated with the manifestation of cardiotoxicity, leading to abnormal cardiac function.

Case presentation A 55-year-old Male was expeditiously transported to our medical facility thirty minutes subsequent to ingesting 10 mL of pyridaben. Upon admission, the patient exhibited symptoms of vomiting and coma. Within the next half-hour, he progressed to hypotonic hypoxemia and hypotension as a result of pyridaben intoxication. With the implementation of prompt gastric lavage, oral administration of activated charcoal, respiratory and circulatory support, as well as continuous renal replacement therapy (CRRT), the patient was ultimately discharged successfully without any lingering sequelae.

Conclusion This case report delineates a rare occurrence of acute human poisoning stemming from contact with novel synthetic pesticides. The document elucidates the clinical manifestations observed and the efficacious administration of supportive therapeutic interventions.

Keywords Pyridaben, Acute respiratory, Circulating failure, Hemoperfusion, CRRT

Background

Pyridaben, a prevalent pyridazinone insecticide employed for safeguarding crops against insects and mites, was investigated by Weidong Qiang. Pyridaben can inhibit mitochondrial complex I, leading to mitochondrial dysfunction that can result in cellular hypoxia, impaired glycolysis, rapid lactic acid accumulation, and the onset of lactic acidosis. Prolonged exposure to pyridaben may elicit toxic effects on neuronal cells. Furthermore, pyridaben can lead to cardiotoxic effects, causing aberrations in cardiac function [6].Pyridaben poisoning, although rare, is associated with high mortality rates. This article presents a case of respiratory and circulatory failure resulting from pyridaben ingestion, yet the patient was ultimately managed successfully.

Case presentation

A 55-year-old Male attempted suicide by ingesting 10 mL of pyridaben, leading to his admission to the emergency department of our hospital 0.5 h post-ingestion. Upon presentation, he exhibited altered mental status, dyspnea, and vomiting coffee-colored liquid, without symptoms such as lacrimation, chest pain, fever, urinary or

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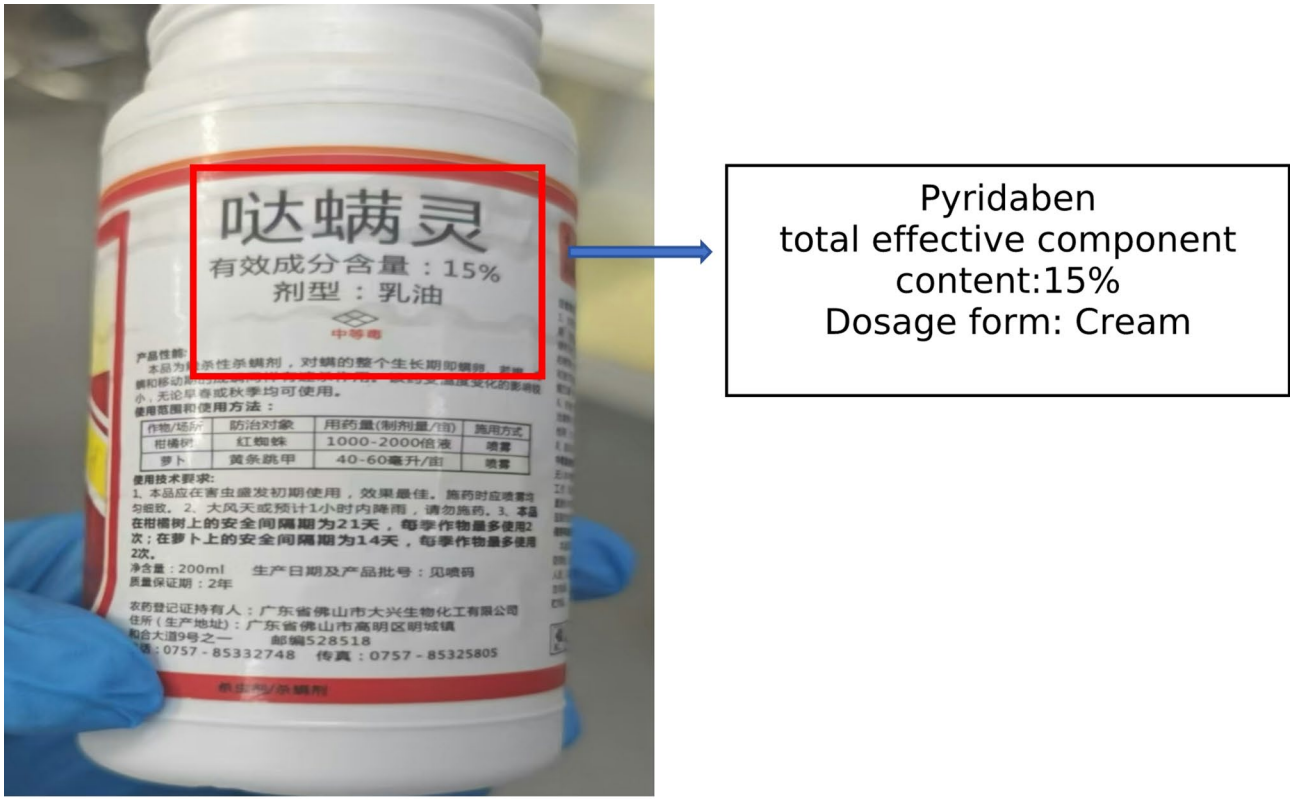
fecal incontinence. There was a distinct smell of poisoning noted.Upon assessment, the patient presented with delirium and exhibited normal pupillary light Reflexes in both eyes, with a diameter of 2.5 mm, and no convulsions or pathological signs. Vital signs showed a heart Rate of 85 beats/min, blood pressure of 95/68 mmHg, and temperature of 36.7 ℃. The patient had a Respiratory rate of 21 breaths/min, and SpO2 levels exceeded 98% without the need for supplemental oxygen. The Results of the blood routine, myocardial Markers, amylase, liver function, renal function, and coagulation tests were within normal limits. Gastric lavage was performed with 10,000 mL of water until clear fluid was obtained.After that, 50 g of activated charcoal was given orally to Remove the remaining toxins from drug use. Intravenous access was established, and intravenous omeprazole 40 mg was administered.



At 17:16, the patient Manifested confusion and experienced a notable decline in blood pressure to 75/47 mmHg. Despite intensive fluid Resuscitation measures, the response was unsatisfactory. Blood oxygen saturation decreased to around 80%. Prompt sedation was provided, followed by orotracheal intubation and initiation of mechanical ventilation [SPN-CPAP, VT 420 mL, PEEP 5 cmH2O, FiO2 60%]. Dopamine was administered intravenously at a Rate of 20 mL/h to increase blood pressure, effectively stabilizing it at approximately 90/60 mmHg and achieving a blood oxygen saturation of 100%. The patient was subsequently transferred to the ICU at 17:45.

In the ICU, blood gas analysis Revealed a pH of 7.039, an HCO3 level of 9.34 mmol/L, a base excess (BE) of −20.59 mmol/L, and a lactate (Lac) level of 15.7 mmol/L. Regarding the biochemical tests, the cardiac troponin I (cTnI) level was 23.9 ng/L, creatinine level was 122 μmol/L, alanine aminotransferase (ALT) level was 73 U/L, and aspartate aminotransferase (AST) level was 164 U/L(Table 1).

The patient received hemoperfusion combined with CRRT using a dual-lumen catheter inserted in the right femoral vein. CRRT was conducted employing intermittent veno-venous hemofiltration (CVVH) for a duration of 48 h, with two sessions of hemoperfusion administered during this period. Simultaneously, the patients was Rehydrated to aid toxin elimination, intravenous administration of diazepam to reduce oxygen consumption, fluid resuscitation and diuresis, along with the Maintenance of internal environmental homeostasis. In

Table 1 The clinical laboratory results of the patient

Time Parameters	0.5 h	3 h	24 h	48 h	36 h
pH(7.35–7.45)	-	7.039	7.163	7.374	-
HCO3(21-27mmol/L)	-	9.34	14.42	28.2	-
-BE(−3-+3)	-	−20.59	−13.38	−2.01	-
Lac(0.5-1.6mmol/L)	-	15.7	10.1	2.0	-
WBC(3.5–9.5 10^9/L)	9.26	10.76	17.17	12.97	4.92
HGB(115–150 g/L)	140	147	162	132	101
PLT(125–350 10^9/L)	258	245	311	159	100
ALT(7-40U/L)	18	73	195	105	61
AST(13-35U/L)	32	164	257	53	37
CREA(41–81μmol/L)	100↑	122	101	74	64
cTnI(0-11pg/ml)	4.0	23.9	537	1177	389

the afternoon of January 10, the patient regained clear consciousness and demonstrated the ability to comply with instructions. Subsequent reevaluation of blood gas analysis showed no deviations from normal parameters. After successful weaning and extubation, the patient's stable vital signs warranted transfer to the nephrology ward. Ultimately, the patient was discharged from the hospital on January 21.

Discussion

Pyridaben is acknowledged for its efficacy as a potent and broad-spectrum pesticide in the control of various phytophagous mites on food plants [1]. Originating from the efforts of Nissan Chemical Industries Ltd in the 1980 s [2], it has evolved into one of the most extensively utilized acaricides in recent decades [3–5]. By the year 2024, China had Registered a total of 353 pyridaben formulations, as reported by the Chinese Pesticide Information Network [6]. The mechanism of action of pyridaben primarily involves the inhibition of muscle and nerve tissues, alongside interference with the synthesis of glutamate dehydrogenase within the electron transport chain of mitochondria, thereby manifesting its insecticidal properties [7]– [8]. Pyridaben demonstrates the capacity to inhibit mitochondrial complex I, leading to mitochondrial dysfunction characterized by cellular hypoxia, impaired glycolysis, rapid lactic acid accumulation, ultimately resulting in lactic acidosis [9]– [10]. Prolonged exposure to pyridaben can elicit toxic repercussions on neuronal cells [11]– [12]. Moreover, pyridaben has been shown to induce cardiotoxic effects, resulting in abnormal cardiac function [13].

In the treatment of acute poisoning, effective antidotes, strategies to diminish poison absorption, facilitation of poison elimination, and prompt life support interventions are pivotal aspects of treatment [14–16]. Currently, there is no specific antidote available for patients suffering from pyridaben poisoning. The primary focus of treatment involves minimizing poison absorption and providing supportive care for organ function. Following oral ingestion of pyridaben, patients May present with altered consciousness, dyspnea, and hypotension approximately 30 min post-exposure Routine blood tests, liver and renal function assessments, electrolyte levels, myocardial enzymes, and pancreatic enzyme levels all yielded normal results. Given the patient's poisoning duration of less than 1 h, the highly toxic yet non-corrosive nature of the poison, and the absence of a specific antidote, the current critical treatment approach involves the removal of unabsorbed poison in the stomach. Gastric lavage and oral activated charcoal administration are essential interventions to mitigate poison absorption [17]– [17]. Following these procedures in the emergency department, the patient subsequently developed coma, Respiratory depression, refractory shock, arrhythmia, and metabolic acidosis after 1 h. Immediate tracheal intubation and fluid resuscitation were promptly initiated to maintain the stability of respiration and circulation. Subsequent laboratory investigations conducted three hours later revealed increased white blood cell count, neutrophil count, and blood glucose levels. Concurrently, elevations in lactic acid levels, transaminase levels, creatinine levels, pancreatic enzyme levels, and troponin levels were observed to varying extents in the patient. Pyridaben, a highly lipid-soluble pesticide, exhibits a propensity to bind to plasma proteins. Following poisoning, it impacts mitochondrial function, consequently leading to a large accumulation of lactic acid. Hemoperfusion, utilizing activated charcoal and resin adsorbents, is employed for the direct removal of toxins from the bloodstream [18]. CRRT is utilized to assist in lactate clearance and rectification of electrolyte imbalances [19]– [20]. Upon immediate admission to the intensive care unit, the patient underwent hemoperfusion to facilitate the Rapid elimination of toxins, thereby mitigating multi-organ damage. After 24 h, the patient's parameters reached their peak levels. Following active blood purification, the patient's poisoning symptoms gradually improved starting from the 48th hour, with all metrics displaying a declining trajectory. Sedative medications were discontinued at 36 h, leading to extubation of the patient, who was subsequently transferred to the general ward for further care.

Conclusion

The successful rescue of the patient is summarized as follows: Firstly, the patient ingested activated charcoal and gastric lavage during the early stages of poisoning, which significantly reduced the absorption of toxins in the digestive tract.Secondly, administering appropriate fluid resuscitation, timely respiratory and circulatory support is essential to ensure adequate tissue perfusion

and oxygen delivery. Thirdly, early initiation of sedation is recommended to prevent convulsions and reduce oxygen consumption. Fourth, timely implementation of blood purification therapies such as hemoperfusion and CRRT is essential for rapid toxin removal and prevention of significant organ damage.

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Authors' contributions
ZD was a major contributor in writing the manuscript.ZWY is involved in the entire process of patient rescue in the emergency department.WJ is responsible for the guidance and correction.All authors reviewed the manuscript.

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Data availability
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Declarations

Ethics approval and consent to participate
Not applicable.

Consent for publication
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Competing interests
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