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CHLORPYRIFOS POISONING: A CASCADE OF CHOLINERGIC CRISIS, ASPIRATION, AND PULMONARY EMBOLISM

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INTRODUCTION

Pesticide poisoning remains a significant public health problem associated with high morbidity and mortality. In Malaysia, agricultural insecticide poisoning is the second most frequent type of pesticide poisoning, with organophosphates (OP), particularly chlorpyrifos, being the leading causative agent.[1]

CASE DESCRIPTION

A 30-year-old man presented to our center 12 hours after intentionally ingesting 50–100 mL of chlorpyrifos-containing termiticide (21.2%) with decreased responsiveness, hypersalivation, and recurrent vomiting. On arrival, his Glasgow Coma Scale (GCS) was E4 V2 M6, with bilaterally 2 mm reactive pupils. Vital signs included a blood pressure of 122/89 mmHg, heart rate of 81 bpm, and SpO_2 of 96% on room air. He exhibited copious foamy oral secretions, generalized muscle weakness, and minimal rhonchi on auscultation. Despite treatment with escalating doses of intravenous atropine and pralidoxime, his persistent vomiting led to aspiration, resulting in respiratory distress requiring mechanical ventilation. He was intubated and maintained on atropine and pralidoxime infusions for 48 hours. Serum cholinesterase testing later returned at 128 U/L (ref: 5320 – 12920 U/L) confirming acute toxicity. He was extubated after three days; however, his hospital course was complicated by a saddle pulmonary embolism, confirmed on CTPA. Tracheal aspirate cultures identified *Klebsiella pneumoniae.*

DISCUSSION

This case highlights the life-threatening sequelae of chlorpyrifos poisoning, including cholinergic crisis and secondary pulmonary embolism. The benefits of atropine in management are well-established. However, the clinical benefits of pralidoxime remain debated, as a recent meta-analysis showed that it does not significantly reduce mortality or ventilator requirements, and may increase the risk of intermediate syndrome.[2] Nevertheless, in the absence of an alternative antidote, pralidoxime remains a mainstay of treatment, especially when administered within 24 to 48 hours of ingestion. For severe toxicity, continuous infusion may offer advantages over intermittent boluses though optimal dosing remains uncertain.[3] The subsequent development of pulmonary embolism may be attributed to prolonged immobilization and a hypercoagulable state induced by systemic inflammation.

CONCLUSION

OP poisoning remains a significant medical emergency. Our case emphasizes the need for monitoring beyond the acute cholinergic phase, as delayed complications can significantly impact patient outcomes. Stricter regulation of public access to hazardous pesticides is essential to prevent similar incidents.

REFERENCES

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