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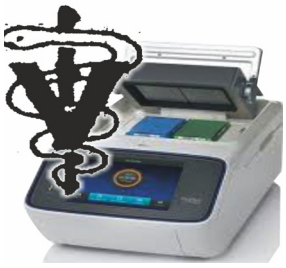

Mass Mortality and Public Health Crisis Following Organophosphate (Sniper®) Poisoning in a Herd of Cattle Grazing in a Cashew (*Anacardium occidentale)* Plantation at Ayigba, Kogi State, Nigeria: A Case Report

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Abstract

A herd of 460 indigenous cattle suffered acute poisoning after grazing a cashew (Anacardium occidentale) plantation where fruits had been sprayed with Sniper® (dichlorvos, an organophosphate insecticide) by the farm owner as a retaliatory measure against recurrent cattle invasion. Within 30 minutes, 70 animals died before veterinary responders arrived. Emergency interventions saved 280 animals, all of which recovered fully. However, several carcasses were later processed and sold for consumption, creating a public health crisis. This report outlines the toxicological mechanism, clinical findings, emergency pharmacological management, and One-Health implications of this catastrophic event.

Keywords: Organophosphate poisoning, Dichlorvos, Cattle, One Health, Nigeria, Toxicology.

1. Introduction

Organophosphate (OP) insecticides remain widely used in sub-Saharan agriculture, notably dichlorvos (2,2-dichlorovinyl dimethyl phosphate), the active ingredient of Sniper®. Dichlorvos which irreversibly inhibits acetylcholinesterase, leading to the accumulation of acetylcholine at cholinergic synapses [16]. In Nigeria, indiscriminate use and repurposing of dichlorvos for non-agricultural applications have led to frequent poisonings in humans and animals [14].

Herders–farmers conflicts in northern Nigeria have occasionally escalated into retaliatory attacks, including deliberate poisoning of grazing livestock [8]. Beyond animal welfare losses, contaminated carcasses pose grave public-health risks when introduced into the food chain. This report documents one such incident, its pharmacological intervention, and the One-Health lessons derived.

2. Case Presentation

2.1 Signalment

- **Species:** Cattle (*Bos indicus*)
- **Breed:** Mixed indigenous
- **Age range:** 8 months- 6 years
- **Number affected:** 460 (total): 70 dead, 280 exposed and treated, 110 unexposed
- **Location:** Outskirts of Anyigba, Dekina LGA, Kogi State, Nigeria
- **Management:** Extensive grazing

2.2 History

According to herders, the cattle had inadvertently grazed within a cashew plantation where fruits had been deliberately treated with Sniper® (dichlorvos). Within minutes of ingestion, exposed animals exhibited marked hypersalivation, generalized muscle tremors, and recumbency, rapidly progressing to collapse. Upon arrival of the veterinary response team approximately 30 minutes post-exposure, seventy carcasses were found at the scene.

2.3 Clinical Findings

- Profuse salivation, miosis, lacrimation, dyspnea, tremors, bradycardia, frequent urination/defecation, and weakness.
- **Temperature:** 39.0–40.4 °C **Pulse:** 60–96 bpm **Respiration:** 40–76 cpm **Mucous membranes:** congested to pale.

2.4 Diagnostic Work-Up

Diagnosis was based on history, clinical signs, and field complete blood count (CBC)/serum results from 10 symptomatic cattle.

Table 1. Mean Haematological profile of poisoned cattle

Parameter	Mean ± s.e.	Reference	Interpretation
PCV (%)	25.8 ± 3.4	30–40	Mild anemia
Hb (g/dL)	8.4 ± 1.2	10–13	Low
WBC (×10³/µL)	14.6 ± 2.3	4–12	Neutrophilic leucocytosis
Neutrophils (%)	78 ± 6	20–45	Neutrophilia
Lymphocytes (%)	16 ± 4	45–75	Lymphopenia
Platelets (×10³/µL)	330 ± 42	200–500	Normal

*Source of Reference values: Jain (1993) [9] and Radostits *et al.* (2007) [15]; s.e.= standard error; n = 10

Table 2. Serum biochemistry and liver function indices (n = 10)

Parameter	**Mean ± SD**	**Reference**	**Interpretation**
AST (U/L)	135 ± 25	60–120	Mild elevation
ALT (U/L)	48 ± 10	20–40	Slight increase
Total protein (g/dL)	6.2 ± 0.5	6–8	Normal
Albumin (g/dL)	2.8 ± 0.3	3–3.5	Slightly low
Serum aceylcholinesterase (U/L)	1,050 ± 240	3,000–6,000	Severely depressed

*Reference values from Kaneko *et al*. (2008) [10] and Radostits *et al*. (2007) [15].

Markedly reduced acetylcholinesterase activity confirmed acute organophosphate exposure.

3. Treatment and Management

Emergency therapy was coordinated by a mobile veterinary team.

- Atropine sulfate 0.5 mg/kg IM, repeated q4h – antimuscarinic counteracting acetylcholine excess.
- Meloxicam 0.5 mg/kg SC SID × 3 days – anti-inflammatory and analgesic (substituted for flunixin once stocks depleted).
- Initial IV administration of 5% dextrose-saline was limited to severely affected or recumbent animals due to logistical constraints.
- Oral rehydration salts (ORS) sachets combined with glucose powder were reconstituted in clean water and administered.
- Activated charcoal (2 g/kg PO) – for animals able to swallow.
- Fluid therapy and hand-feeding for recumbent cattle under shade.

4. Therapeutic Outcome

Out of a total of 460 cattle, 70 were dead on arrival, 280 recovered fully after supportive therapy, and 110 were unexposed. No relapse was recorded after 10 days.

5. Complications and Public-Health Implications

Despite veterinary advice to incinerate carcasses, some were transported to local markets. Consumption of such meat poses serious risk as dichlorvos residues persist post-cooking [4]. Chronic human exposure may cause neurotoxicity and endocrine disruption. The event triggered local conflict among herders, farmers, and authorities, demonstrating the intertwined animal–human–environment interface, central to One Health.

6. Discussion

The history of organophosphate (Sniper®) use on the farm and the rapid onset of signs involving profuse salivation, miosis, dyspnea, and tremors together with markedly depressed serum cholinesterase levels confirmed the diagnosis of acute dichlorvos (organophosphate) toxicity. These pathognomonic features align with the biochemical mechanism of organophosphate compounds causing irreversible inhibition of acetylcholinesterase (AChE), resulting in the accumulation of acetylcholine at neuromuscular junctions and continuous stimulation of cholinergic receptors [16].

The haematological profile showed mild anaemia and stress leukogram characterized by neutrophilia and lymphopenia, consistent with acute stress and systemic inflammation following toxic insult. The biochemical pattern revealed elevated AST and ALT values, indicating hepatocellular injury and leakage of marker enzymes possibly due to hypoxia/secondary oxidative stress. Depressed serum cholinesterase served as the diagnostic hallmark of organophosphate exposure. Similar findings were described by Ngowi *et al*. (2007) [13] and Adesokan *et al*. (2020) at parasympathetic effector sites. The absence of oxime reactivators such as pralidoxime, a standard adjunct in OP therapy was a limitation from field logistics challenges. Nevertheless, the early atropinization significantly reduced mortality, consistent with the findings of Peter and Cherian (2015) [16] that timely atropine remains the single most life-saving intervention in OP toxicity.

The use of meloxicam (0.5 mg/kg SC SID for 3 days) as the preferred NSAID was justified on pharmacokinetic and safety grounds. Although flunixin meglumine provides a broader anti-endotoxin profile [12], meloxicam's selective COX-2 inhibition, extended half-life, and reduced gastrointestinal adverse effects make it ideal for field therapy in ruminants [5]. Diclofenac and piroxicam, though available, were avoided due to their narrow therapeutic margins in stressed livestock. Thus, meloxicam provided a sustained analgesic support while maintaining favorable pharmacodynamic safety.

The IV administration of 5% dextrose-saline ensured hydration and maintained perfusion during shock [15]. Activated charcoal was used in animals that could still swallow, serving as a toxin adsorbent [17]. Supportive nursing care, including hand-feeding and protection from direct sunlight, was essential for recovery [18].

The survival of 280 animals despite massive exposure underscores the efficacy of prompt field response and the pharmacological soundness of the chosen regimen. Comparable herd-level interventions reported by Ogunseitan (2021) [14] and Adesokan *et al*. (2020) [2] demonstrated that mortality can be significantly reduced when antidotal and supportive care are instituted within 1 hour of exposure.

Beyond clinical management, this episode exposes broader One Health failures in Nigeria's agro-ecosystem. The use of dichlorvos as a tool for retaliation reflects weak pesticide regulation, inadequate public awareness, and escalating land-use conflicts. The subsequent processing and sale of poisoned carcasses for human consumption raised a serious food safety and toxicological crisis, since dichlorvos residues are

[2] in field reports of pesticide-associated livestock deaths in sub-Saharan Africa.

Therapeutic intervention hinged on atropine sulfate, an established antidote for organophosphate poisoning. Its use at 0.5 mg/kg IM every 4 hours effectively counteracted muscarinic overstimulation by competitively inhibiting acetylcholine at parasympathetic effector sites. The absence of oxime reactivators such as pralidoxime, a standard adjunct in OP therapy was a limitation from field logistics challenges. Nevertheless, the early atropinization significantly reduced mortality, consistent with the findings of Peter and Cherian (2015) [16] that timely atropine remains the single most life-saving intervention in OP toxicity.

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Although dichlorvos is thermolabile and undergoes rapid hydrolysis, the safety implication of cooking contaminated carcasses remains complex and should be discouraged. The compound degrades to metabolites such as dimethyl phosphate, dichloroacetaldehyde, and dichloroethanol, along with demethylated oxidative derivatives formed during metabolism and heating [3,9]. While regulatory reviews indicate these hydrolysis products are generally less potent acetylcholinesterase inhibitors than the parent molecule [3], several, particularly dichloroacetaldehyde and dichloroethanol, exhibit distinct toxicological profiles, including mucosal irritation, hepatotoxicity, and potential genotoxicity [4,14].

Residue analyses have shown that cooking and frying may reduce intact dichlorvos but do not consistently eliminate residues or metabolites owing to variable tissue penetration and incomplete degradation [6]. Consequently, reliance on culinary heat treatment as a detoxification measure is unreliable, and the sale or consumption of carcasses exposed to dichlorvos contamination constitutes a significant public-health hazard, warranting laboratory residue/metabolite testing and stricter regulatory oversight [3].

This event mirrors earlier reports of pesticide-related cattle deaths in Tanzania [13] and chlorpyrifos-poisoned sheep in Egypt [1], emphasizing that indiscriminate pesticide use remains a continental problem requiring cross-sectoral One Health surveillance.

Conclusion

The incident highlights the deadly intersection of agrochemical misuse, farmer–herder conflict,

and food-chain safety lapses. Prompt pharmacological intervention saved most animals, but the public-health fallout underscores the urgency for stricter pesticide regulation, community education, and an integrated One-Health framework.

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