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ORGANOPHOSPHATE POISONING ADMISSIONS IN A RURAL HOSPITAL: A TWO-YEAR RETROSPECTIVE STUDY

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Abstract

Organophosphorus pesticides remain a leading cause of poisoning admissions in rural hospitals, and most deaths follow deliberate self-ingestion. This short narrative review summarises the published literature relevant to hospital admission series: the epidemiology of pesticide poisoning in agricultural communities, the clinical syndromes and severity markers, the evidence behind atropine, oximes, decontamination and supportive care, and the effect of pesticide regulation on mortality. Outcomes depend on the compound, dose, solvent, delay to treatment and the availability of ventilatory support. Many treatments rest on weak trial evidence, and published hospital series are heterogeneous. The article reports no new patient data and does not analyse any hospital's records.

1. INTRODUCTION

Pesticide poisoning is a quiet public health emergency of farming communities. Estimates from systematic reviews place pesticide ingestion among the most important methods of suicide worldwide, accounting for a substantial share of all suicides in low- and middle-income countries, and organophosphorus compounds have been a major contributor in much of South Asia and parts of Africa and Latin America [1, 2, 3]. Many of these deaths happen in or on the way to small district or rural hospitals that were never designed to deliver intensive care.

Organophosphates act by inhibiting acetylcholinesterase, the enzyme that breaks

down acetylcholine at synapses. The resulting excess of the transmitter overstimulates muscarinic and nicotinic receptors and the brain, producing a recognisable but variable clinical picture [4, 5]. The compounds were developed from the chemistry of nerve agents in the middle of the twentieth century, and thousands of tonnes remain in agricultural use in some countries because they are cheap and effective [6, 7].

Retrospective admission series from rural hospitals are a common form of publication on the subject. They typically count admissions over a year or two, describe age, sex, intent, the compound, time to arrival, treatment and outcome, and report mortality. Such series are valuable for local planning, but they are hard to compare, and they are sometimes read as if they could answer questions about treatment efficacy. This review therefore sets out the wider literature against which any such series should be read [4, 8, 9].

Poor data compound the problem. In many countries where organophosphate poisoning is common, vital registration is incomplete, deaths at home are not certified by a physician and causes are miscoded. Estimates of national burden therefore depend on hospital series, police records and modelling, and reviewers have repeatedly cautioned that official statistics undercount pesticide deaths [2, 3, 10]. A nationally representative survey in India, for example, found suicide mortality considerably higher than official figures suggested [10]. Local admission audits are among the few sources that can describe the problem at community level.

The review reports no data from any hospital. It draws on published trials, systematic reviews and cohort studies, and it states numerical findings only where they are widely reported. Sections follow the sequence a clinician would meet: who is admitted, what the poisoned patient looks like, what treatment is supported by evidence, and what could prevent the admissions in the first place.

2. EPIDEMIOLOGY IN RURAL SETTINGS

Global estimates of fatal pesticide self-poisoning suggest that a large proportion of cases occur in the Western Pacific and South-East Asia regions, with the highest case counts reported from countries where farming is the main livelihood and storage of pesticides at home is common [2, 3]. Within countries, rural districts carry the burden, since pesticides are at hand in homes and fields and the hospital is far away. The proportion of cases that are suicidal rather than occupational or accidental is high in hospital series from Asia [1, 2, 10].

Typical patients in rural series are young adults, with a roughly even sex distribution or a slight excess of men, and women outnumber men in some populations. Poisoning is often impulsive. Interviews with survivors in several countries have described acts taken within minutes of an argument or an episode of acute distress, using whatever was available in the household, which is the reason that access to the poison is so important [1, 6, 11]. Seasonal peaks coincide with spraying and sowing seasons, and with periods of financial stress for farmers.

Social and economic circumstances shape the pattern. Debt, crop failure, family conflict, alcohol use and untreated depression are repeatedly named in interviews and psychological autopsy studies as precipitants of pesticide self-poisoning [1, 10, 11]. Access to mental health services is limited in the same rural areas where pesticide availability is greatest. Admission figures should therefore be read as a marker of distress in a community as well as of a toxicological hazard, and an intervention that addresses only the hospital phase will not change the underlying demand [1, 6].

Occupational and accidental poisoning are less common in hospital series, but they are probably under-represented because mild cases are treated at home or at primary care clinics. Agricultural workers may be exposed through skin contact when mixing concentrates, leaking sprayers or poorly maintained protective equipment, and the effects are usually less severe than after ingestion [4, 5, 12]. Children are exposed by accident when pesticides are stored in drink bottles or left within reach.

Case fatality in hospital series varies widely between settings, and between compounds. A prospective cohort in Sri Lanka showed that fatality after self-poisoning with different agricultural pesticides ranged from low to very high, with the compound explaining much of the difference [9, 13]. Within organophosphates, the same group found that dimethyl and diethyl compounds, and those with particular solvent formulations, caused different clinical patterns and mortality [14, 15]. An audit that reports a single case-fatality figure, without identifying compounds, is therefore of limited use for comparison.

3. CLINICAL PICTURE AND SEVERITY

Classical acute poisoning shows a cholinergic crisis. Muscarinic features include small pupils, sweating, salivation, lacrimation, bronchorrhoea, bronchospasm, vomiting, diarrhoea and a slow heart rate; nicotinic effects include fasciculations, weakness and paralysis; central effects include agitation, confusion, seizures and coma [4, 5, 16]. Respiratory failure is the usual cause of death in the first day, owing to bronchorrhoea, bronchoconstriction, central respiratory depression and paralysis of respiratory muscles [16, 17].

Not every patient follows the textbook. Heart rate may be fast rather than slow, as nicotinic effects on sympathetic ganglia compete with vagal stimulation, and pupils may be normal or dilated in mixed presentations, particularly if the patient has also ingested other substances such as alcohol [4, 16, 18]. Diagnostic mistakes occur when clinicians rely on a single sign. The garlic-like odour of some compounds, the smell of hydrocarbon solvents on the clothes and a history of ingestion offered by relatives often prove more informative than pupil size [4, 18].

The intermediate syndrome was described in 1987 in patients who, after apparently recovering from the cholinergic crisis, developed weakness of neck flexors, proximal limb muscles and respiratory muscles 24 to 96 hours after ingestion [5, 19]. It reflects persistent neuromuscular junction dysfunction and may require ventilation for days. The syndrome is more commonly recognised after exposure to highly lipophilic compounds, and its cause is debated. A related, much rarer complication is delayed polyneuropathy, which appears two to three weeks after exposure to certain compounds and is linked to inhibition of neuropathy target esterase [5, 12, 19].

Severity grading helps to allocate scarce resources. Scoring systems such as the Peradeniya organophosphorus poisoning scale, the poisoning severity score and the Glasgow Coma Scale have been compared in studies of admitted patients; the Glasgow Coma Scale and the poison severity score both predicted the need for ventilation and death, with reasonably good discrimination [20, 21]. Simple measures, including respiratory rate, oxygen saturation and level of consciousness at admission, are available in even the smallest hospital and carry much of the prognostic information [17, 21].

Laboratory tests are limited in rural hospitals. Red cell acetylcholinesterase and plasma butyrylcholinesterase activities support the diagnosis and are widely used in research, but turnaround is slow, the assays are not standardised, and treatment decisions should not wait for them [4, 12]. Butyrylcholinesterase recovers faster and is less closely tied to synaptic function, so serial values are sometimes misleading. Clinical judgement remains the basis for decisions in most settings.

Carbamate insecticides, which also inhibit cholinesterase but reversibly, can produce a similar syndrome that is usually shorter and less severe, and distinguishing them from organophosphates at the bedside is difficult [5, 18, 22]. The distinction matters mainly for oxime use, since reactivation is of little value in carbamate poisoning and oximes have been thought to be potentially harmful in some compounds such as carbaryl. In practice, rural clinicians seldom know with certainty which compound was ingested, which is a further reason for simple protocols built around observable signs [4, 16, 18].

Compound-specific differences deserve emphasis. In a prospective cohort, patients poisoned with highly toxic dimethyl compounds such as fenthion died from early

respiratory failure or later from the intermediate syndrome, whereas diethyl compounds like chlorpyrifos produced a pattern in which deaths were more often delayed and associated with prolonged cholinergic toxicity [13, 14]. Solvent and formulation add to the picture, since hydrocarbon solvents may contribute to aspiration pneumonitis and to some of the toxicity attributed to the active ingredient [15].

Other complications also feature in series. Aspiration pneumonia is common because of vomiting and reduced consciousness, hospital-acquired infection follows prolonged ventilation, pancreatitis and hyperglycaemia occasionally occur, and cardiac arrhythmias including QT prolongation have been described [4, 12, 16]. These complications add to the workload of staff in small hospitals and should be anticipated.

4. MANAGEMENT AND EVIDENCE

Evidence on treatment, outcome determinants, clinical features and prevention is gathered in table 1, and the distribution of the entries across those groups is shown in fig. 1. The table makes one point plainly: the number of well-supported statements about prevention and severity is higher than the number about pharmacological rescue. It reflects a research record in which cohort studies and policy analyses, mostly from South Asia, have produced firmer findings than the few randomised trials of antidotes [4, 8, 11, 23].

The first priorities are the ones that apply to any poisoned patient: airway, breathing and circulation. Oxygen, suction and early intubation for patients with bronchorrhoea or falling consciousness may save lives before any antidote is given. Staff should wear gloves and aprons while undressing and washing the patient, since contaminated clothing can poison carers [4, 16]. Removal of clothes, washing of skin and hair with soap and water, and the avoidance of unnecessary delay for investigations are the foundation of decontamination.

Atropine is the mainstay of treatment and the only antidote with universal support. It blocks muscarinic receptors and reverses bronchorrhoea, bronchospasm and bradycardia, but has no effect on nicotinic paralysis [4, 22, 24]. The aim is to dry the chest secretions, restore a heart rate above about 80 beats per minute, and produce a systolic blood pressure above 80 mmHg with clear lungs, instead of aiming at dilated pupils or a dry mouth [4, 25]. Large doses may be needed, in the order of tens of milligrams on the first day in severe cases, and over-atropinisation with delirium and hyperthermia is a recognised hazard.

Speed of atropinisation matters. A systematic comparison of recommended regimens found a wide range in how quickly different protocols achieved control, and suggested that a regimen of doubling the dose every five minutes until the target is reached would attain control faster than slower schedules [25]. The protocol is simple enough to be taught to nurses. In many rural hospitals the real barrier is a shortage of atropine or reluctance to give large doses because of unfamiliarity, and not lack of knowledge about the pharmacology [4, 16, 25].

Oximes are intended to reactivate the inhibited enzyme before it ages, a process in which the phosphorylated enzyme becomes resistant to reactivation. Their value is a long-standing controversy. A systematic review of trials found conflicting results, and the Cochrane review concluded that the evidence was insufficient to say whether oximes help or harm, because the trials were small and used different drugs, doses and patient groups [26, 27]. A meta-analysis found no clear benefit and raised the possibility of harm [28], and a large randomised trial of pralidoxime at the World Health Organization recommended dose in Sri Lanka showed no survival benefit, although subgroup questions remained open [23].

One randomised trial in India reported that continuous infusion of pralidoxime gave better outcomes than intermittent bolus doses [29], but it had methodological limits and was not reproduced. Laboratory work suggests that benefit depends on the compound, since some inhibited enzymes age within minutes and others take much longer [24, 30]. Practical advice therefore varies, from

avoiding oximes to using them early in selected patients, and many hospitals follow local guidelines in the absence of firm evidence [4, 16, 24].

Table 1: Selected published findings on organophosphate poisoning relevant to hospital admission series

Topic	Reported finding	Ref.
Cholinergic crisis Early syndrome	Muscarinic, nicotinic and central features; respiratory failure is the usual early cause of death.	[4, 16, 17]
Intermediate syndrome	Neck, proximal limb and respiratory muscle weakness one to four days after the crisis.	[5, 19]
Compound differences	Toxicity and clinical pattern vary markedly between organophosphates and formulations.	[13, 14, 15]
Severity scoring	Glasgow Coma Scale and poison severity score predict ventilation and death.	[20, 21]
Atropine regimens	Rapid dose-doubling achieved control faster than slower regimens in a comparison of protocols.	[25]
Oximes	Trials and reviews inconclusive; large trial showed no survival benefit.	[23, 26, 27, 28]
Multiple-dose activated charcoal	No mortality reduction in a large randomised trial of self-poisoning.	[31]
Gastric lavage	Hazards including aspiration documented in a resource-poor setting.	[32]
Pesticide regulation in Sri Lanka	Bans of hazardous compounds followed by falls in suicide and pesticide deaths.	[33, 34]
Systematic review of restrictions	Restrictions on highly hazardous pesticides linked to fewer suicides in most settings.	[11]

Gut decontamination has fared poorly in trials. A large randomised trial of multiple-dose activated charcoal in self-poisoning, in which organophosphates formed a major group, showed no reduction in mortality, and work on gastric lavage has pointed to hazards of the procedure, including aspiration, in resource-poor settings [31, 32]. Single-dose charcoal may still be offered to alert patients who arrive within an hour, though evidence of benefit is weak. Induced vomiting should be avoided [4, 31, 32].

Supportive care ultimately determines survival. Ventilatory support, sedation for agitation, treatment of seizures with benzodiazepines, management of aspiration and careful fluid balance are all part of the work, and prolonged mechanical ventilation may be needed in the intermediate syndrome [4, 16, 17]. The respiratory-failure study reported that survival depended on prompt intubation and ventilation and that many deaths occurred before arrival at an intensive care unit [17]. Transfer to a referral centre is a central question for a rural hospital, and in some regions the shortage of ventilators drives mortality as much as the toxicity itself.

Timing of transfer deserves a clearer statement than it usually receives. A patient who is stable, conscious and responding to atropine may be better kept in the receiving hospital until the intermediate syndrome window has passed, while a deteriorating patient needs a ventilator before they reach one. Published guidance suggests initiating treatment before transfer, ensuring the patient is accompanied by a trained nurse with atropine and suction, and communicating the compound and doses given to the receiving team [4, 16, 17]. Accounts from rural districts describe journeys of several hours, during which unrecognised respiratory failure is the commonest cause of avoidable death [17].

5. PREVENTION AND PRACTICE

The strongest evidence for reducing deaths concerns restriction of the most hazardous pesticides. Sri Lanka banned the most toxic organophosphates in stages from the 1980s and 1990s, and national suicide rates fell afterwards, with analyses attributing a large number of averted deaths to the bans without evidence of method substitution on the same scale [33, 34]. A systematic review of national regulations in several countries found that restrictions on highly hazardous pesticides were associated with fewer pesticide suicides and fewer suicides overall in most of the evaluated settings [11].

Regulation relies on classification of hazard. The World Health Organization classification

of pesticides, which ranks them by acute oral and dermal toxicity, is widely used to decide which should be restricted or withdrawn [35]. Proposals for a minimum list of pesticides for developing countries, using agronomic need and low toxicity as criteria, have been put forward as a pragmatic approach where banning everything is not feasible [7]. Lethality data from clinical series, like those in Sri Lanka, give a human basis for such lists in addition to animal tests [9, 13].

Evidence on specific interventions at village level is accumulating. Community-based projects that provide lockable storage containers to farming households have been proposed, and reviews of means-restriction strategies conclude that reducing availability at the point where impulsive acts occur is among the most promising suicide-prevention measures available to rural communities [6, 11]. Retailers can be trained not to sell the most hazardous products to people who appear distressed, though evidence of effect is weaker than for regulation [6, 11].

Community measures complement regulation. Safe storage in locked containers away from the home, education for farmers on handling and spraying, and responsible retailing have been explored; the evidence for household storage interventions is encouraging but limited [6, 11]. Mental health care, crisis support and reduction of alcohol misuse are further elements, since a large share of self-poisoning follows an acute crisis in a person with distress that could be treated [1, 10]. Hospitals have a role as well, since survivors of self-poisoning are at risk of further attempts and need follow-up.

6. LIMITATIONS AND FUTURE DIRECTIONS

The literature has several shortcomings. Randomised trials are few and small, most patient series are retrospective single-centre accounts with incomplete records, and definitions of severity and outcome vary. Mortality figures from rural hospitals depend on referral patterns, transport times and the willingness of families to leave patients in hospital, so cross-site comparison is unreliable [8, 9, 10]. Publication bias may favour hospitals with better data, and under-reporting is probably worst where the problem is greatest.

A word on generalisation is also needed. Findings from Sri Lanka, where much of the high-quality cohort and trial work has been done, may not transfer to other regions with different compounds, formulations, hospital structures and referral patterns. The pesticide market changes, and substitutes for banned compounds, including less toxic organophosphates and other chemical classes, carry their own risks that need monitoring [9, 13, 35]. Conclusions about any individual hospital's admissions require local data, and this review cannot supply them.

Future work should record the compound, dose, time to treatment and initial severity in a standard way, and combine hospital data with community and forensic data to estimate the true burden. Pragmatic multicentre trials of oximes, atropine regimens and new antidotes are needed, and so are studies of how best to deliver basic life support in small hospitals [4, 24, 27]. Evaluation of regulation and means-restriction programmes should continue in new settings [6, 11, 34].

7. CONCLUSION

Organophosphate poisoning in rural hospitals is a problem of deliberate self-harm with an exceptionally lethal method. Case fatality is determined by the compound, dose, delay and available care, and a hospital series that omits those details cannot be interpreted [9, 13, 14]. Clinical recognition, rapid atropinisation and ventilatory support are the elements most likely to save lives in a small hospital [4, 16, 17, 25].

Evidence for oximes, decontamination and adjunct therapies is weaker than many guidelines imply, and trials in this field remain few [23, 26, 27, 31]. The best-supported intervention is prevention, through regulation of highly hazardous pesticides and safer storage, which has been followed by large falls in deaths [11, 33, 34]. Admission series should be read in that light.

8. ABBREVIATIONS

AChE-Acetylcholinesterase, OP-Organophosphorus, WHO-World Health Organization, GCS-Glasgow Coma Scale,

PSS-Poison severity score, ICU-Intensive care unit, PAM-Pralidoxime, mmHg-Millimetres of mercury

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10. AUTHORS CONTRIBUTIONS

All the authors have contributed equally.

11. CONFLICT OF INTERESTS

Declared none

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