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# Changing spectrum of acute poisoning in North India: A hospital-based descriptive study

Ashok Kumar Pannu, Ashish Bhalla\*, Vitla Vamshi, Manish Kumar Upadhyay, Navneet Sharma, Susheel Kumar

Department of Internal Medicine, Postgraduate Institute of Medical Education and Research, Chandigarh, India

\*Corresponding author

## Abstract:

**OBJECTIVES:** Evaluating local trends and continued monitoring of patterns of acute poisoning are essential for prompt recognition of the toxidromes, the establishment of immediate treatment facilities (e.g., antidote availability), and effective preventive strategies (e.g., governmental regulation on hazardous substances marketing). We aimed to describe the prevalence of the various types of poisoning and associated case fatality in our academic hospital in North India.

**METHODS:** A prospective observational descriptive study was conducted, enrolling patients aged  $\geq 13$  years with acute poisoning for 17 months from December 2016 to December 2017 and from September 2019 to December 2019, for a total of 17 months.

**RESULTS:** Four hundred and two patients were enrolled (median age 28 years; 63.2% males). Majority of the acute poisoning cases resulted from ingestion ( $n = 391$ , 97.3%) and the primary intention was most commonly self-harm ( $n = 314$ , 78.1%). The major types of poisoning were pesticide ( $n = 264$ , 65.7%), drug overdose ( $n = 77$ , 19.2%), and corrosive ingestion ( $n = 31$ , 7.7%). Pesticides included insecticides ( $n = 146$ , 36.3%; cholinesterase inhibitors,  $n = 91$ ), fungicides ( $n = 76$ , 18.9%; all aluminum phosphide), herbicides ( $n = 22$ , 5.5%; paraquat,  $n = 19$ ), and rodenticides ( $n = 20$ , 5.0%; coumarin-derived substances,  $n = 12$ ). Benzodiazepines ( $n = 33$ ) and opioids ( $n = 25$ ) were frequent causes of drug overdose. 95.3% ( $n = 379$ ) received preliminary treatment at the previous health-care center, including gastric lavage ( $n = 239$ ) and antidotes ( $n = 73$ ). In-hospital case fatality rate was 17.3% ( $n = 58$ ).

**CONCLUSION:** Herbicide ingestion and opioid overdose are emerging threats with a gradual decline in organophosphate and aluminum phosphide poisoning. Despite improving management of acute poisoning, the overall case fatality rate remains substantial.

## Keywords:

Acute poisoning, case fatality, drug overdose, herbicide, opioid, paraquat, pesticide

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## ORCID:

AKP: 0000-0002-4476-3478

AB: 0000-0001-5210-1012

VV: 0000-0002-5725-2204

MKU: 0000-0002-1374-2279

NS: 0000-0001-5707-9686

SK: 0000-0003-1660-7686

## Address for correspondence:

Dr. Ashish Bhalla,  
4<sup>th</sup> Floor, F Block, Nehru  
Hospital, Postgraduate  
Institute of Medical  
Education and Research,  
Chandigarh - 160 012,  
India.  
E-mail: [bhalla.chd@gmail.com](mailto:bhalla.chd@gmail.com)

## Introduction

Acute poisoning is of major concern because of the potential for rapid deterioration and fatal outcomes in previously healthy individuals. The patients frequently present to the emergency department (ED) and often require admission to critical care units. Worldwide, pesticide

ingestion remains a common form of acute poisoning, accounting for at least one in seven self-poisoning and about two hundred thousand deaths annually.<sup>[1,2]</sup> Because low-middle-income countries (LMIC), especially in the Asia-Pacific region, predominantly have agriculture-based economies, easy accessibility of pesticides results in a high incidence of poisoning.<sup>[3-5]</sup> In India, organophosphate and aluminum

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### Box-ED

#### What is already known on the study topic?

Evaluating local trends and continued monitoring of patterns of acute poisoning are essential for prompt recognition of the toxidromes, the establishment of immediate treatment facilities, and effective preventive strategies

Pesticide ingestion is the main form of acute poisoning in agricultural communities such as North India.

#### What is the conflict on the issue? Has it importance for readers?

The studies addressing acute poisoning trends in North India are scarce and have not been regularly updated

A paradigm shift in the poisoning spectrum is expected in our region, given improved agricultural practices, the opioid epidemic, advancement in critical care, and enhanced governmental regulation on pesticides in the recent past.

#### How is this study structured?

This was a single-center, prospective observational descriptive study conducted with 402 patients admitted to the ED of an academic hospital in North India.

#### What does this study tell us?

Herbicide ingestion and opioid overdose are emerging threats with a gradual decline in organophosphate and aluminum phosphide poisoning

Despite improved treatment at the primary health centers, the overall case fatality rate remains substantial.

phosphide are the most common pesticides that cause acute poisoning.<sup>[5-11]</sup> Household products such as disinfectants or cleaners remain the continuous source of intentional or unintentional exposures.<sup>[9-12]</sup> In recent years, worldwide reports have established an increasing incidence of a drug overdose, including pharmaceutical agents (e.g., antidepressants) or illicit drugs (e.g., opioids).<sup>[2,6,13,14]</sup>

Although acute poisoning is a universal phenomenon, the spectrum varies in different parts of the world and even within a country.<sup>[2,5-12]</sup> Socioeconomics, cultural environment, and toxicant availability are the essential factors influencing the poisoning spectrum and trends. Evaluating local trends and continued monitoring of poisoning patterns is critical for prompt recognition of the toxidromes, establishing immediate treatment facilities at the primary health centers (e.g., antidote availability), and effective preventive strategies (e.g., awareness campaigns, governmental restrictions on hazardous substances marketing).

The studies addressing poisoning trends in India, especially in the northern states, are scarce and have not been regularly updated.<sup>[5,9-11,15-18]</sup> The marked change in the

acute poisoning spectrum is expected in our region, given improved agricultural practices, the opioid epidemic, advancement in critical care management (e.g., ventilator beds availability), and enhanced governmental regulation on pesticides in the recent past. Therefore, we prospectively examined acute poisoning cases at our tertiary care hospital in North India in the current study to describe the poisoning spectrum and the associated case fatality ratio (CFR). The main aim was to gain further knowledge about trends in the prevalence of the various types of poisoning compared with our center's previous data and address their optimal management.

## Methods

### Study design

This prospective observational descriptive study was conducted at the ED of a tertiary care hospital and an apex referral center in North India. About 200 patients present daily to the ED triage of this 1948-bedded hospital from a large population of North India.<sup>[19]</sup> About 100–120 are admitted to ED, and toxicological emergencies constitute 1–2 cases per day.<sup>[19,20]</sup> The age criteria for admission to our adult medical emergency is  $\geq 13$  years. Our study combines two prospectively collected data from December 2016 to December 2017 and from September 2019 to December 2019 (17 months). The institutional ethics committee approved the study (No. INT/IEC/2017/1450, date March 10, 2016; INT/IEC/2018/1116, date March 18, 2018). Written informed consent was obtained from all study patients or a legally authorized representative (if the patient could not consent). There was no funding source or financial support for the study.

### Study population

The patients aged  $\geq 13$  years were recruited based on a history of toxin exposure and/or typical clinical manifestations of acute toxicity. Patients with snake bites, chronic toxicity or drug toxicity at therapeutic doses, or cases brought dead were not included.

### Data collection

Sociodemographic features (age, sex, marital status, occupation, educational status, and residence), route of exposure (i.e., ingestion, inhalation, or dermal absorption), the intention of poisoning (i.e., self-harm or suicidal, accidental, or homicidal), type of the intoxicant (pesticide, drug overdose, corrosive, etc.), clinical features, and laboratory abnormalities were recorded. Toxidrome-specific investigations such as plasma cholinesterase levels and urine drug screen (benzodiazepines, barbiturates, cocaine, opiates, tetrahydrocannabinol, and amphetamine) were performed when judged to be appropriate. We recorded the time of toxin exposure, treatment received at the

previous health-care center, and the time elapsed before arrival at the first medical center and admission to our ED. The cases were divided into five major types of acute poisoning-pesticide, drug overdose, corrosive ingestion, miscellaneous, and unknown. Pesticides included four groups-insecticide, fumigant, herbicide, and rodenticide.

### Management

Toxicity management followed standard institutional protocols. All the patients received primary emergency medical care addressing the airway, breathing, and circulation at ED admission. If the patient was brought to the hospital within the first 1–2 h of toxin ingestion and after the airway was secured, gastric lavage using normal saline was performed for all cases except corrosive, petroleum products, and paraquat ingestions. In cholinesterase inhibitor poisoning, atropinization was performed according to the doubling-dose method, followed by an atropine infusion for maintenance.<sup>[3]</sup> Flumazenil was indicated only in pure benzodiazepine overdose in a nonhabituated user for reversal of conscious sedation.<sup>[21]</sup> The opiate antagonist naloxone was given in suspected or confirmed opioid overdose. Other antidotes used were folinic acid (leucovorin) for methotrexate overdose, N-acetyl cysteine for drug-induced acute liver failure (e.g., in yellow phosphorus, zinc phosphide, or methotrexate toxicity), Vitamin K for severe coagulopathy (e.g., in coumarin rodenticide toxicity), sodium bicarbonate for tricyclic antidepressant-related cardiac toxicity, and methylene blue or high-dose ascorbic acid for methemoglobinemia (e.g., in naphthalene toxicity).<sup>[22-24]</sup> Patients with corrosive ingestion, petroleum product ingestion or inhalation, and toxic gases exposure were treated with supportive care without steroids.<sup>[25-27]</sup> Endoscopy was performed for caustic ingestions, ideally within 12 h and preferably not later than 24–48 h of ingestion.<sup>[25]</sup> Based on the standard staging system, the endoscopic evaluation of the severity of a corrosive esophageal injury was done.<sup>[25]</sup>

Initially, the patients were admitted to an ED observation unit (a high dependency unit) for high-quality supportive care. Subsequently, they were shifted to an intensive care unit, a step-down unit, or a general medical ward based on the clinical severity of the patients. They were followed till discharge or death during hospitalization. The outcomes were documented as death, discharge (planned) or leave against medical advice, and length of stay. In-hospital CFR was calculated after excluding the patients who left against medical advice.

### Statistical analysis

We used the Statistical Package for the Social Sciences (SPSS Inc., Chicago, IL, USA) for statistical analysis. The categorical data were recorded as

frequency (*n*) and percentage (%). The continuous variables were described using mean  $\pm$  standard deviation or median with interquartile range (IQR), depending on whether the data were normal in distribution. The Kolmogorov–Smirnov test checked the normalcy of data. The Chi-square test was used to determine if there was a difference between the categorical variables. A two-sided  $P \leq 0.05$  was considered statistically significant for all statistical tests.

## Results

### Sociodemographic characteristics

Four hundred and two cases of acute poisoning were identified. The median age was 28 years (IQR, 22–38; range, 13–79). The peak incidence occurred in the third decade ( $n = 153$ , 38.1%), and about 80% ( $n = 320$ ) of the study population was younger than 40 years. Table 1 shows the sociodemographic profile of the patients.

**Table 1: Sociodemographic profile of the study patients (n=402)**

Parameter	Frequency, n (%)
Age groups (years)	
13-19	67 (16.7)
20-29	144 (35.8)
30-39	96 (23.9)
40-49	50 (12.4)
50-59	26 (6.5)
$\geq 60$	19 (4.7)
Gender	
Male	254 (63.2)
Female	148 (36.8)
Marital status	
Married	231 (57.5)
Unmarried or single	166 (41.3)
Unknown	5 (1.2)
Residence	
Rural	264 (65.7)
Urban	138 (34.3)
Education status	
No formal education	122 (30.3)
Primary school	124 (30.8)
Secondary or high school	90 (22.4)
College or university	37 (9.2)
Unknown	29 (7.2)
Occupation	
Farmer	109 (27.1)
Housewife	89 (22.1)
Student	74 (18.4)
Private-sector employee	52 (12.9)
Government employee	11 (2.7)
Driver	7 (1.7)
Businessman	5 (1.2)
Daily-wage worker	5 (1.2)
Miscellaneous	8 (2.0)
Unemployed	42 (10.4)

The majority of the patients developed toxicity after ingestion ( $n = 391, 97.3\%$ ), while inhalation ( $n = 6, 1.5\%$ ) and dermal exposure ( $n = 5, 1.2\%$ ) were uncommon. The distribution of exposure to poison remained similar among different age groups (13–19 years, 20–39 years,  $\geq 40$  years) and both genders. The intention of poisoning is shown in Figure 1. The intention (self-harm versus accidental) was similar in males (77.8% vs. 22.2%) and females (79.7% vs. 20.3%). Accidental exposure was more in adolescents (13–19 years) and young adults (20–39 years) frequently had self-harm ingestion [Table 2]. Alcohol consumption before acute poisoning was present in 5.0% of patients.

### Primary treatment before admission

The median time to reach the first medical center after toxin exposure was 1.0 h (IQR, 0.5–3.5; range 0.16–168.0). Before admission to our ED, the median time elapsed was 7.5 h (IQR, 3.5–26.7; range, 0.5–672.0). 95.3% ( $n = 379$ ) received preliminary care at the previous health-care center, where the poisoning treatment given was gastric lavage ( $n = 239$ ) and specific antidote administration ( $n = 73$ ), including atropine ( $n = 67$ ), flumazenil ( $n = 3$ ), naloxone ( $n = 2$ ), and N-acetyl cysteine ( $n = 1$ ).

### The spectrum of acute poisoning

The prevalence of various types of acute poisoning is demonstrated in Table 3. Pesticides comprised about two-thirds of the cases, with cholinesterase inhibitors (22.6%), aluminum phosphide (18.9%), and paraquat (4.7%) being the common compounds. Drug overdose ( $n = 77, 19.2\%$ ) and corrosive ingestion ( $n = 31, 7.7\%$ ) remained prevalent. Plasma cholinesterase levels were done in 39 cases of cholinesterase inhibitors poisoning and were detected low in 38. Urine drug screen was positive in 19 out of 39 cases and detected opioids ( $n = 7$ ), benzodiazepine ( $n = 4$ ), tetrahydrocannabinol ( $n = 3$ ), barbiturate ( $n = 1$ ), both benzodiazepine and barbiturate ( $n = 2$ ), and both benzodiazepine and opioids ( $n = 2$ ). The poisoning spectrum was similar among males and females [Figure 2]. Table 4 shows the distribution of acute poisoning in different age groups of the study. Except for a slight increase in herbicide poisoning cases, the poisoning trend did not change between the two study periods [Figure 3].

**Table 2: Intention of poisoning in different age groups of the study**

Intention of poisoning	Age 13-19 years ( $n=67$ ), $n$ (%)	Age 20-39 years ( $n=240$ ), $n$ (%)	Age $\geq 40$ years ( $n=93$ ), $n$ (%)	$n$
Self-harm intention	46 (68.7)	197 (82.1)	71 (76.3)	0.052
Accidental	21 (31.3)	43 (17.9)	22 (23.7)	

### Clinical presentation of common toxidromes

Cholinesterase inhibitor poisonings ( $n = 91$ ) invariably presented with a cholinergic crisis. The usual features were respiratory distress ( $n = 76$ ), vomiting ( $n = 72$ ), excessive salivation or sweating ( $n = 59$ ), miosis ( $n = 58$ ), altered sensorium (52), muscle weakness ( $n = 43$ ), abdominal pain ( $n = 34$ ), and seizure ( $n = 7$ ). The common manifestations of aluminum phosphide poisoning ( $n = 76$ ) were vomiting ( $n = 61$ ), shock (26), and altered mentation ( $n = 23$ ). All patients with paraquat poisoning ( $n = 19$ ) developed multiorgan

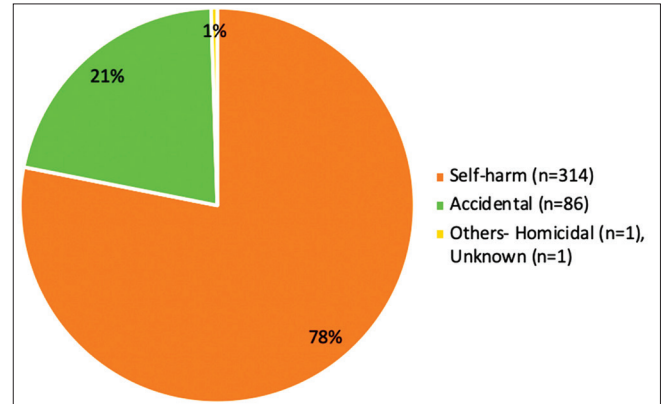


Figure 1: Intentions of the poisoning in the study population ( $n = 402$ )

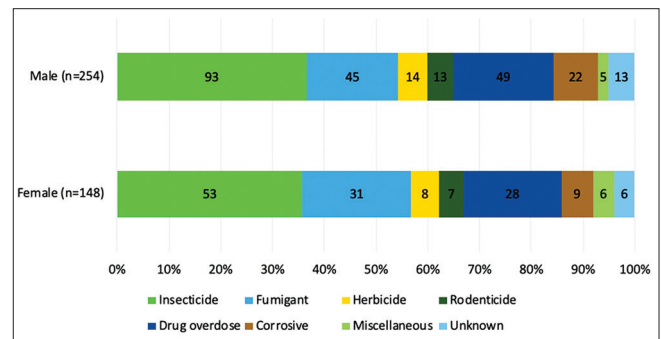


Figure 2: Distribution of acute poisoning among males ( $n = 254$ ) and females ( $n = 148$ )

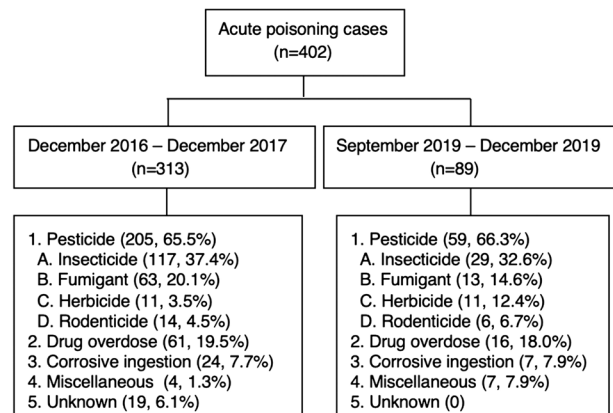


Figure 3: Types of poisoning in the two study periods

**Table 3: Spectrum of acute poisoning and associated case fatality (n=402)**

Compounds	Frequency, n (%)	Case fatality <sup>a</sup> , n (%)
Pesticides	264 (65.7)	29 (13.3)
Insecticides	146 (36.3)	13 (8.8)
Cholinesterase inhibitors	91 (22.6)	8 (10.0)
Organophosphate <sup>b</sup>	39	3 (8.6)
Carbamate	1	0
Unidentified	51	5 (12.8)
Insect repellants	17 (4.2)	0
Amitraz	16	0
DEET	1	0
Pyrethroids	4 (1.0)	0
Cypermethrin	2	0
Deltamethrin	2	0
Miscellaneous	4 (1.0)	1 (25.0)
Neonicotinoid (Imidacloprid)	2	0
Boric acid	1	1 (100)
Organochlorine	1	0
Unidentified	30 (7.5)	4 (26.1)
Fumigants	76 (18.9)	8 (12.9)
Aluminum phosphide	76	8 (12.9)
Herbicides	22 (5.5)	6 (37.5)
Paraquat	19	6 (42.9)
2,4-Dichlorophenoxyacetic acid	1	0
Butachlor	1	0
Unidentified	1	0
Rodenticides	20 (5.0)	2 (13.3)
Coumarin-derived substances	12	1 (8.3)
Zinc phosphide	6	1 (16.7)
Yellow phosphorus	2	0
Drug overdose	77 (19.2)	13 (20.6)
Benzodiazepine	33	6 (20.7)
Opioid	25	4 (21.1)
Tricyclic antidepressant	8	1 (12.5)
Methotrexate	5	1 (20.0)
Antiepileptic drug	2	1 (50.0)
Cannabis	2	0
Cough syrup	1	0
Unidentified	1	0
Corrosive ingestion	31 (7.7)	5 (18.5)
Acids <sup>c</sup>	24	4 (19.0)
Phenyle (phenol)	7	1 (16.7)
Miscellaneous <sup>d</sup>	11 (2.7)	1 (11.1)
Unknown	19 (4.7)	10 (76.9)
Total	402	58 (17.3)

<sup>a</sup>Case fatality (%) was calculated after excluding the cases who left against medical advice, <sup>b</sup>Organophosphates include dichlorvos (*n*=22), monocrotophos (*n*=5), profenofos (*n*=4), chlorpyrifos (*n*=4), phorate (*n*=2), triazophos (*n*=1), and dimethoate (*n*=1), <sup>c</sup>Acids include household cleaning products containing inorganic acids such as hydrochloric acid (e.g., Harpic™) or sulfuric acid, <sup>d</sup>Miscellaneous compounds include petroleum products (*n*=3), naphthalene ball (*n*=2), mushroom poisoning (*n*=1), mercury tablet (*n*=1), button battery (*n*=1), glass powder (*n*=1), copper sulfate (*n*=1), and carbon monoxide (*n*=1). DEET: Diethyltoluamide

dysfunction, i.e., two or more organs injury, including oropharyngeal mucosa ulceration, jaundice, renal failure, or acute hypoxemic respiratory failure. Acute ingestion of amitraz (*n* = 16) frequently resulted in neurological

abnormalities with altered sensorium (*n* = 13) or seizure (*n* = 2).

Benzodiazepines overdose (*n* = 33) usually presented with altered mentation (*n* = 13), vomiting (*n* = 11), seizure (*n* = 4), and dyspnea (*n* = 4). Altered sensorium (*n* = 17), vomiting (*n* = 7), dyspnea (*n* = 3), and seizure (*n* = 2) were present in patients with opioid intoxication (*n* = 25). Twenty four of 31 cases of corrosive ingestion underwent endoscopy, demonstrating esophageal injury of grade 0 (*n* = 1), grade 1 (*n* = 1), grade 2a (*n* = 11), grade 2b (*n* = 6), grade 3a (*n* = 4), and grade 3b (*n* = 1).

### Outcomes

In-hospital CFR was 17.3% (*n* = 58), and 278 (82.7%) patients survived. Sixty-nine patients left against medical advice. CFR associated with the various intoxicants is shown in Table 3. The median duration of hospitalization was 2 days (IQR, 1–4). Only 13.7% (*n* = 55) had a stay of 7 days or more.

### Discussion

This study demonstrated a shift in the spectrum of acute poisoning at our academic institute in North India. The proportion of intentional self-harm cases has significantly increased. While pesticide poisoning with organophosphate and aluminum phosphide has gradually trended downward, the herbicide paraquat has emerged as a lethal toxin. Drug overdose remained common with a growing incidence in opioid cases. Corrosive agents and other readily available household products accounted for a significant number. Most patients received preliminary treatment at previous healthcare centers. CFR remained high, especially with paraquat, drug overdose, and corrosive ingestion.

The primary route of poisoning remained ingestion for self-harm. The proportion of intentional self-harm to accidental exposure (about 4:1) has significantly increased compared to the previous data.<sup>[5,11]</sup> It further supports the burgeoning suicidal poisoning in our geographic region.<sup>[15]</sup> Concurring the previous reports, young adults, male gender, married status, agricultural job, low education level, and rural background were at high risk.<sup>[5,11,15-18]</sup> Increased stress at the workplace, higher family expectations, peer pressure, or lack of job options might be responsible for their high vulnerability to self-poisoning. Although the adolescents had more unintentional exposures, a self-poisoning suicide attempt in about 70% of this age group is significant.

Pesticides remained the most common type of acute poisoning, with significant contribution from organophosphate and aluminum phosphide, albeit

less prevalent than our previous data [Table 5].<sup>[5,11]</sup> The potential explanation for this gradual decline in these pesticides is governmental regulations on selling World health Organization hazard class Ia organophosphates (e.g., methyl parathion, phorate) or replacing 3-g tablets of aluminum phosphide with granules or powder (lesser toxic formulations).<sup>[5,28]</sup> A reduction in organophosphate and aluminum phosphide proportions in tertiary care or referral centers might have resulted from an improvement in medical care in primary health centers. In contrast to our 2009 data, most study patients arrived at our ED after receiving primary care in peripheral centers, including poisoning treatments (e.g., gastric lavage, atropine).<sup>[11]</sup> Moreover, the time elapsed to reach the first medical center after exposure was significantly less (1 h vs. 3 h).<sup>[11]</sup> Despite a global fall in incidence and mortality, pesticide poisoning with organophosphate or aluminum phosphide is a significant problem in agriculture-dependent economies of South East Asia, China, and Africa.<sup>[1-5,29-33]</sup> Apart from advancements in critical care, the specific management of these toxidrome remains almost the same for many decades. Many novel treatments have been evaluated to improve outcomes;

however, they remain controversial or necessitate further research to confirm their efficacy.<sup>[8,34-36]</sup>

Although the annual global manufacturing of herbicides outweighs insecticides several-fold, human herbicide poisoning is uncommon and considered less critical than insecticide poisoning, especially in LMIC.<sup>[1-5,37]</sup> Indian data on herbicide poisoning are limited and primarily based on case reports.<sup>[5-11,38-40]</sup> However, a never-before incidence of about 6% of herbicide ingestion (with paraquat being the most prevalent) was detected in our study. These results are critically important because clinicians in these areas may not be familiar with the varied presentations of herbicide toxicity and organ involvement, resulting in misdiagnosis or delayed diagnosis.<sup>[38-40]</sup> For example, paraquat poisoning typically manifests with jaundice, renal failure, and respiratory distress with pulmonary infiltrates on chest radiography, mimicking common medical emergencies, such as severe pneumonia, sepsis, or tropical illnesses (e.g., malaria, leptospirosis, scrub typhus, or dengue).<sup>[39]</sup>

In contrast to the LMIC, drug overdose remains the main form of acute poisoning in western countries.<sup>[2,6,13,14]</sup> The Centers for Disease Control and Prevention has recently reported an increasing incidence of drug overdose and related deaths in the United States.<sup>[41,42]</sup> Our study detected that nearly one-fifth of acute poisoning cases were attributed to drug overdose, higher than seen (about 10%) in India's recent extensive toxicological data.<sup>[6]</sup> Overall, benzodiazepines remained on top; however, the progression of the global opioid epidemic in North India has led to a sharp increase in opioid overdose cases in our ED, which was less prevalent previously.<sup>[5,6,9,11]</sup> We found that <10% of the patients with opioid overdose received naloxone in primary health centers, which might be due to no ready availability of the drug, the lack of knowledge of its use, or the unfamiliarity of the toxicity features.

**Table 4: Distribution of acute poisoning in different age groups of the study**

Compounds	Age 13-19 years (n=67), n (%)	Age 20-39 years (n=240), n (%)	Age ≥40 years (n=95), n (%)
Pesticide	48 (71.6)	154 (64.2)	62 (65.3)
Insecticide	36 (53.7)	77 (32.1)	33 (34.7)
Fumigant	5 (7.5)	48 (20.0)	23 (24.2)
Herbicide	3 (4.5)	15 (6.3)	4 (4.2)
Rodenticide	4 (6.0)	14 (5.8)	2 (2.1)
Drug overdose	14 (20.9)	44 (18.3)	19 (20.0)
Corrosive ingestion	2 (3.0)	23 (9.6)	6 (6.3)
Miscellaneous	3 (4.5)	5 (2.1)	3 (3.2)
Unknown	0	14 (5.8)	5 (5.3)

**Table 5: Trends in the types and case-fatality rate of acute poisoning at our center**

Study	Murali et al. <sup>[5]</sup>	Mittal et al. <sup>[11]</sup>	Index study
Type of study	Retrospective	Prospective	Prospective
Duration	1990-2004	2009	2016-19 (17 months)
Total cases (n)	2884	102	402
Anti-cholinesterase, n (%)	1011 (35)	27 (27)	91 (23)
Aluminum phosphide, n (%)	739 (26)	20 (20)	76 (19)
Herbicide, n (%)	NA	0	22 (6)
Benzodiazepine, n (%)	NA	6 (6)	33 (8)
Opioids, n (%)	NA	2 (2)	25 (6)
Corrosive, n (%)	NA	10 (10)	31 (8)
Case fatality (%)	16	19	17

NA: Not available data

Corrosive ingestion with household cleaners was common, although with a lesser frequency than in recent studies from the urban areas of North India.<sup>[9,12]</sup> Unintentional cleaner ingestion frequently occurs because of its similar physical appearance to water.<sup>[12]</sup> Similarly, methotrexate toxicity typically results from suprathreshold ingestion (repeated daily dosing), given oral formulations of methotrexate (Folitrax™ is a familiar brand) and folic acid (a common supplementation with methotrexate) are “look-alike sound-alike” medications.<sup>[22]</sup> Acetaminophen overdose remains the leading cause of acute liver failure in North America, Europe, and Australia due to its widespread use and easy accessibility; however, it has been detected infrequently in the reports from LMIC such as India, which is further supported by our data.<sup>[5,6,11-18,43]</sup>

Despite continued efforts to counteract the trend and advancements in emergency and critical care management of acute poisoning, case fatality remained substantial [Table 5].<sup>[5,11]</sup> Organophosphate and aluminum phosphate-related CFR showed a gradual decline; however, an increase in paraquat poisoning diluted an overall improvement in pesticide-related CFR. Due to its easy availability, rapid-onset severe toxicity, and no specific antidote, paraquat is emerging as a lethal toxin in our area. Global data have demonstrated a drastic reduction in poisoning-related CFR after banning such highly toxic pesticides by policy actions or legal mechanisms.<sup>[28,44-47]</sup> Fatal drug overdoses are also a significant concern in our center, requiring urgent strategies such as wide distribution of naloxone and identifying high-risk patients receiving antidepressant, antipsychotic, or opioid medications.

### Limitations

Although this study had a prospective analysis with a reasonable sample size, the limitations were single-center data and a tertiary-care hospital referral bias. The generalizability of our results requires confirmation from multicenter, more extensive studies in India. We also caution its generalizability to specific age groups. Given the study design and unavailability of many laboratory parameters, a detailed statistical analysis (e.g., CFR predictors) could not be performed.

### Conclusion

This study captures the changing trends of acute poisoning in North India. While the prevalence of organophosphate and aluminum phosphide poisoning gradually declines, herbicide ingestion and opioid overdose are emerging threats. Despite improvement in the treatment at the primary health centers and advancements in critical care management, overall CFR from acute poisoning remains substantially high. These results highlight the need for tailored policy interventions such as restricting the sale of lethal toxins like paraquat and wide distribution of naloxone in this geographic region.

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### Author contributions statement

AKP: Conceptualization (supporting); Formal analysis (lead); Writing-original draft (lead); Writing-review and editing (lead).

AB: Conceptualization (lead); Methodology (lead); Writing-review and editing (supporting).

VV: Data curation (lead); Formal analysis (supporting).

MKU: Data curation (supporting); Formal analysis (supporting); Writing-original draft (supporting).

NS: Conceptualization (supporting); Methodology (supporting).

SK: Conceptualization (supporting); Methodology (supporting).

The corresponding author is responsible for ensuring that the descriptions are accurate and agreed upon by all authors.

### Conflicts of interest

None declared.

### Ethical approval

The Institutional Ethics Committee of Postgraduate Institute of Medical Education and Research, Chandigarh (India) approved the study (No. INT/IEC/2017/1450, date March 10, 2016).

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## References

1. Mew EJ, Padmanathan P, Konradsen F, Eddleston M, Chang SS, Phillips MR, *et al.* The global burden of fatal self-poisoning with pesticides 2006-15: Systematic review. *J Affect Disord* 2017;219:93-104.
2. WHO and Food and Agriculture Organization of the United Nations. Preventing Suicide: A Resource for Pesticide Registrars and Regulators. Geneva: World Health Organization; 2019. Available from: <https://apps.who.int/iris/bitstream/handle/10665/326947/9789241516389-eng.pdf>. [Last accessed on 2021 May 03].
3. Pannu AK, Bhalla A, Vishnu RI, Garg S, Dhibar DP, Sharma N, *et al.* Cardiac injury in organophosphate poisoning after acute ingestion. *Toxicol Res (Camb)* 2021;10:446-52.
4. Pannu AK, Bhalla A, Sharma A, Sharma N. "PGI Score": A simplified three-point prognostic score for acute aluminum phosphide poisoning. *Indian J Crit Care Med* 2020;24:790-3.
5. Murali R, Bhalla A, Singh D, Singh S. Acute pesticide poisoning: 15 years experience of a large North-West Indian hospital. *Clin Toxicol (Phila)* 2009;47:35-8.
6. Mittal C, Singh S, Kumar-MP, Varthya SB. Toxicoepidemiology of poisoning exhibited in Indian population from 2010 to 2020: A systematic review and meta-analysis. *BMJ Open* 2021;11:e045182.
7. Peshin SS, Srivastava A, Halder N, Gupta YK. Pesticide poisoning trend analysis of 13 years: A retrospective study based on telephone calls at the National Poisons Information Centre, All India Institute of Medical Sciences, New Delhi. *J Forensic Leg Med* 2014;22:57-61.
8. Anbalagan LC, Arora N, Pannu AK. Management of acute aluminum phosphide poisoning: Has anything changed? *Drug Metab Lett* 2021;14:106-16.
9. Mathew R, Jamshed N, Aggarwal P, Patel S, Pandey RM. Profile of acute poisoning cases and their outcome in a teaching hospital of north India. *J Family Med Prim Care* 2019;8:3935-9.
10. Ramesha KN, Rao KB, Kumar GS. Pattern and outcome of acute poisoning cases in a tertiary care hospital in Karnataka, India. *Indian J Crit Care Med* 2009;13:152-5.
11. Mittal N, Shafiq N, Bhalla A, Pandhi P, Malhotra S. A prospective observational study on different poisoning cases and their outcomes in a tertiary care hospital. *SAGE Open Med* 2013;1:2050312113504213.
12. Peshin SS, Gupta YK. Poisoning due to household products: A ten years retrospective analysis of telephone calls to the National Poisons Information Centre, All India Institute of Medical

- Sciences, New Delhi, India. *J Forensic Leg Med* 2018;58:205-11.
13. McHugh RK, Nielsen S, Weiss RD. Prescription drug abuse: From epidemiology to public policy. *J Subst Abuse Treat* 2015;48:1-7.
  14. Gummin DD, Mowry JB, Spyker DA, Brooks DE, Beuhler MC, Rivers LJ, *et al.* 2018 annual report of the American Association of Poison Control Centers' national poison data system (NPDS): 36<sup>th</sup> annual report. *Clin Toxicol (Phila)* 2019;57:1220-413.
  15. Singh D, Dewan I, Pandey AN, Tyagi S. Spectrum of unnatural fatalities in the Chandigarh zone of north-west India – A 25 year autopsy study from a tertiary care hospital. *J Clin Forensic Med* 2003;10:145-52.
  16. Thomas M, Anandan S, Kuruvilla PJ, Singh PR, David S. Profile of hospital admission following acute poisoning experiences from major teaching hospital in south India. *Adverse Drug React Toxicol Rev* 2000;19:313-7.
  17. Singh S, Sharma BK, Wahli PL, Anand BS, Chugh KS. Spectrum of acute poisoning in adults (10 year experience). *J Assoc Physicians India* 1984;32:561-3.
  18. Sharma BR, Harish D, Sharma V, Vij K. Poisoning in northern India: Changing trends, causes and prevention thereof. *Med Sci Law* 2002;42:251-7.
  19. Pannu AK, Saroch A, Kumar M, Behera A, Nayyar GS, Sharma N. Quantification of chronic diseases presenting in the Emergency Department and their disposition outcomes: A hospital-based cross-sectional study in north India. *Trop Doct* 2022;52:276-9.
  20. Saini MK, Kumar H, Saini K, Behera A, Pannu AK, Soundappan K, *et al.* Impact of lockdown on medical emergency visits during the COVID-19 pandemic in India. *Postgrad Med J* 2022;98:e112-4.
  21. Penninga EI, Graudal N, Ladekarl MB, Jürgens G. Adverse events associated with flumazenil treatment for the management of suspected benzodiazepine intoxication – A systematic review with meta-analyses of randomised trials. *Basic Clin Pharmacol Toxicol* 2016;118:37-44.
  22. Pannu AK. Methotrexate overdose in clinical practice. *Curr Drug Metab* 2019;20:714-9.
  23. Bradberry SM, Thanacoody HK, Watt BE, Thomas SH, Vale JA. Management of the cardiovascular complications of tricyclic antidepressant poisoning: Role of sodium bicarbonate. *Toxicol Rev* 2005;24:195-204.
  24. Pannu AK, Singla V. Naphthalene toxicity in clinical practice. *Curr Drug Metab* 2020;21:63-6.
  25. Tovar R, Leikin JB. Irritants and corrosives. *Emerg Med Clin North Am* 2015;33:117-31.
  26. Bolla T, Kanneganti V, Aggarwal T, Singh S, Chaudhary S, Pannu AK. Fire-breather's lung: Hydrocarbon pneumonitis. *Postgrad Med J* 2022;98:e50.
  27. Pannu AK, Kumar M, Bhalla A. Hazardous chemical emergencies and poisonings. *N Engl J Med* 2019;381:392-3.
  28. Peter JV, Jerobin J, Nair A, Bennett A. Is there a relationship between the WHO hazard classification of organophosphate pesticide and outcomes in suicidal human poisoning with commercial organophosphate formulations? *Regul Toxicol Pharmacol* 2010;57:99-102.
  29. Buckley NA, Fahim M, Raubenheimer J, Gawarammana IB, Eddleston M, Roberts MS, *et al.* Case fatality of agricultural pesticides after self-poisoning in Sri Lanka: A prospective cohort study. *Lancet Glob Health* 2021;9:e854-62.
  30. Pannu AK, Bhalla A, Vishnu RI, Dhibar DP, Sharma N, Vijayvergiya R. Organophosphate induced delayed neuropathy after an acute cholinergic crisis in self-poisoning. *Clin Toxicol (Phila)* 2021;59:488-92.
  31. Pannu AK, Bhalla A. A simple tool predicts mortality in aluminum phosphide self-poisoning. *Indian J Crit Care Med* 2020;24:755-6.
  32. Pannu AK. Pulmonary management in aluminum phosphide poisoning. *Indian J Crit Care Med* 2017;21:63-4.
  33. Pannu AK, Jhuria L, Bhalla A, Sharma N. PGI score: Prospective validation and correlation with SOFA, SAPS-II, and APACHE-II scores for predicting outcomes in acute aluminum phosphide poisoning. *Toxicol Res (Camb)* 2022;11:361-6.
  34. Pannu AK, Garg S, Bhalla A, Dhibar DP, Sharma N. Lipid emulsion for the treatment of acute organophosphate poisoning: An Open-Label randomized trial. *Clin Toxicol (Phila)* 2022;60:602-8.
  35. Kumar HM, Pannu AK, Kumar S, Sharma N, Bhalla A. Magnesium sulfate in organophosphorus compound poisoning: A prospective open-label clinician-initiated intervention trial with historical controls. *Int J Crit Illn Inj Sci* 2022;12:33-7.
  36. Pannu AK, Bhalla A, Gantala J, Sharma N, Kumar S, Dhibar DP. Glucose-insulin-potassium infusion for the treatment of acute aluminum phosphide poisoning: An open-label pilot study. *Clin Toxicol (Phila)* 2020;58:1004-9.
  37. EPA United States Environmental Protection Agency. Pesticides Industry Sales and Usage 2008–2012 Market Estimates. Washington DC: U.S. Environmental Protection Agency; 2017. Available from: [https://www.epa.gov/sites/default/files/2017-01/documents/pesticides-industry-sales-usage-2016\\_0.pdf](https://www.epa.gov/sites/default/files/2017-01/documents/pesticides-industry-sales-usage-2016_0.pdf). [Last accessed on 2020 Nov 16].
  38. Pannu AK, Saroch A, Agrawal J, Sharma N. 2,4-D poisoning: A review with illustration of two cases. *Trop Doct* 2018;48:366-8.
  39. Pannu AK, Veerabhadraiah A, Patel I. Commentary on: Chen F, Ye Y, Jin B, Yi B, Wei Q, Liao L. Homicidal paraquat poisoning. *J Forensic Sci* 2019;64:963-4.
  40. James N, Bakshi R, Rudresh SS, Kaushik K, Ghumaan KS, Pannu AK. Pneumoperitoneum from pneumomediastinum in paraquat poisoning. *Trop Doct* 2021;51:241-2.
  41. Centers for Disease Control and Prevention. Increase in Fatal Drug Overdoses Across the United States Driven by Synthetic Opioids Before and During the COVID-19 Pandemic. Atlanta: CDC; 2020. Available from: <https://emergency.cdc.gov/han/2020/han00438.asp>. [Last accessed on 2021 Feb 08].
  42. Vivolo-Kantor AM, Seth P, Gladden RM, Mattson CL, Baldwin GT, Kite-Powell A, *et al.* Vital signs: Trends in emergency department visits for suspected opioid overdoses – United States, July 2016–September 2017. *MMWR Morb Mortal Wkly Rep* 2018;67:279-85.
  43. Chiew AL, Buckley NA. Acetaminophen poisoning. *Crit Care Clin* 2021;37:543-61.
  44. Cha ES, Chang SS, Gunnell D, Eddleston M, Khang YH, Lee WJ. Impact of paraquat regulation on suicide in South Korea. *Int J Epidemiol* 2016;45:470-9.
  45. Mann JJ, Apter A, Bertolote J, Beautrais A, Currier D, Haas A, *et al.* Suicide prevention strategies: A systematic review. *JAMA* 2005;294:2064-74.
  46. Yip PS, Caine E, Yousuf S, Chang SS, Wu KC, Chen YY. Means restriction for suicide prevention. *Lancet* 2012;379:2393-9.
  47. Barber CW, Miller MJ. Reducing a suicidal person's access to lethal means of suicide: A research agenda. *Am J Prev Med* 2014;47:S264-72.