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ISSN: 2663-9491 e-ISSN: 2789-6897

ORIGINAL ARTICLE

A Clinico-Epidemiological Study of Pyrethroid Poisoning

DOI: 10.5281/zenodo.18014019

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Received: 03 Dec 2025
Accepted: 08 Dec 2025
Published: 17 Dec 2025

Published by:
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ABSTRACT

Introduction: *Bangladesh, with its agriculture-based economy, has long been known to have a high incidence of poisoning from agrochemical compounds. These are mainly organophosphorus, carbamate, and pyrethroid poisoning. There is also a recent trend of increased use of non-organophosphorus compounds like pyrethroids.* ***Methods & Materials:*** *A descriptive observational study was carried out in the all-medicine wards of Chattagram Medical College Hospital from May 2017 to April 2018. A total of 222 cases fulfilled the criteria of the case definitions of agro-chemical poisonings. Out of them, only 38 cases were confirmed as Pyrethroid Poisoning cases. Data were recorded from the day of admission up to the date of discharge by the investigator herself.* ***Result:*** *By sample identification, 38 cases were confirmed as pyrethroid poisoning. Most patients were aged 12–19 years (mean age 25.34 ± 10.83) and were mainly farmers or students (each 29%). Patients from outside Chattogram had a significantly longer ingestion-to-admission time than those from within the city (p=0.006). All presented with nausea and hypersalivation, commonly with abdominal pain. Laboratory abnormalities included neutrophilic leukocytosis (13 cases), hyperglycemia (5), elevated ALT (6), and raised creatinine (3). Among 35 cases with documented outcomes, 33 (94.3%) improved.* ***Conclusion:*** *Synthetic Pyrethroids after oral ingestion can produce significant morbidities and rare cases of mortalities, when patients were treated late in the hospital, and also if there was the presence of any comorbidities like stroke, hypertension, and respiratory failure.*

Keywords: *Pyrethroids, Poisoning, Epidemiology, Toxicity*

(The Insight 2025; 8(3): 577-582)

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INTRODUCTION

Pesticide poisoning is a common method of suicide attempt and, less commonly, accidental poisoning in developing countries [1]. Based on types of pests controlled, pesticides include insecticides, herbicides, acaricides, fungicides, rodenticides etc. [2]. Until 2013, 2894 different types of pesticides were approved in Bangladesh [2]. Organophosphate compounds (OPCs) are the most frequently reported pesticides used for poisoning in Bangladesh [3]. Very few non-OPCs (Pyrethroids) are mentioned as poisoning agents. The inappropriate use of pesticides is common in developing countries, as they are used in agriculture, where rural people live and work in proximity to these compounds, which are often stored in and around the home. According to the WHO, the estimated mortality of poisoning in India was 10.3 deaths per 100,000 population, whereas in Bangladesh it was 1.1 deaths per 100,000 population [4]. WHO also reported that the global frequency of total pesticide poisoning was 30% (range 27-37%) [5]. In South East Asia, it was 20.7% by Gunnel et al. in 2007 [4]. Another study in South India (Mysore) showed that 39% of total poisoning was due to insecticides, among them OPC 71%, Pyrethroids 7% and Carbamates 6.5%. In Bangladesh, pesticide poisoning accounted for 39% (CI 37.6-40.6) of total poisoning cases [6]. Reported frequency of different pesticides includes: OPC 89.8%, Rodenticides 4.3%, Carbamate 4%, Unknown 1.6%, Pyrethroids 0.5% [6]. Previously, in our setting, all patients with a history of ingestion of an agrochemical compound were labeled as OPC poisoning and received management of OPC poisoning, including antidotes like atropine and pralidoxime, sometimes leading to serious complications and unnecessary treatment burden. Clinical features, complications, and management of the Pyrethroid poisoning are different from those of OPC poisoning. Due to a lack of awareness among caregivers, these patients are not being identified and managed properly. A recently done pilot study in Chattogram Medical College Hospital revealed that, when patients or attendants are specifically asked, the pesticide name could be identified in 69% of cases. The name of the pesticide could be determined by asking patients to bring the offending bottle to the hospital, sending a photo message of the bottle, or retrieving the name by phone call. Comparison of methodologies used in Bangladesh suggests that Toxidromic identification (identification done by signs and symptoms of patients) possibly overestimates OPC poisoning. In a study where the container was identified, OPCs were found in 78.1%, carbamates in 6.2% and pyrethroids in 15.6% cases [3]. In addition, clinical differentiation between mild OPC poisoning and Pyrethroid poisoning can be difficult in the acute phase (both can cause constriction of the pupil and increased secretions) [8]. These further stresses the importance of early identification and determination of demographic status and

ISSN: 2663-9491 e-ISSN: 2789-6897

clinical outcome of Pyrethroid poisoning. A study on demographic characteristics of pesticide-poisoned cases showed that the highest number of patients belonged to the age group of 21-30 years (38.1%), followed by patients aged under 20years (33.8%) [3]. Marital status of the respondents showed that most of the patients were unmarried (56.4%) [4]. Occupation of the studied cases was reported that 18.3% of them were students, 16.7% housewives, 11.7% businessmen, 11.6% farmers, and 5.7% others [3]. In another study, 47% were farmers, 16% students, and 13% were housewives [3]. Regarding place of residence, the majority, 80% of the respondents, lived in rural areas [4]. Regarding clinical features shown by the patients, a previous study reported that Pyrethroid ingestion gave rise within minutes to sore throat, nausea, vomiting, abdominal pain, mouth ulceration, increased salivation, and dysphagia [9]. An epidemiological study of Acute Pyrethroid poisoning among cotton farmers in China showed that vomiting was common, followed by dizziness, headache, and fatigue [10]. There were fewer frequent palpitations, chest tightness and blurred vision, coma, and convulsions [10]. Pulmonary complications were reported, including aspiration pneumonia and pulmonary oedema [11]. Cases with developing muscle fasciculation and convulsion showed occasional abnormal reports of EMG and EEG [12]. Cardiac events reported in acute Pyrethroid poisoning included ST and T wave changes, sinus tachycardia, ventricular ectopic, and rarely sinus Bradycardia [13]. These changes resolved over 2-14 days. Abnormal laboratory findings following ingestion of Pyrethroids included leukocytosis, metabolic acidosis, increased creatinine, increased AST activity, and most of the patients recovered within 6 days [14].

METHODS & MATERIALS

This descriptive observational study was conducted in the all-medicine wards of Chattagram Medical College Hospital from May 2017 to April 2018. A total of 222 patients met the criteria for agrochemical poisoning, among which 38 cases were confirmed as pyrethroid poisoning. Data were collected prospectively from the day of admission until discharge by the investigator. Convenient sampling was used. All poisoning cases admitted to the medicine ward were first recorded in a registration book, and screening for pyrethroid poisoning was performed according to predefined case definitions. After identifying confirmed cases, data were obtained on sociodemographic characteristics, clinical features, complications, laboratory findings, treatments received, follow-up results, outcomes, and causes of death. Clinical signs and symptoms were recorded at admission and monitored daily until discharge or death. The clinical course of each patient was evaluated through daily follow-up and relevant laboratory investigations. Statistical analysis was performed using SPSS version 20 (IBM Corp., Armonk, NY, USA). A p-value of less than 0.05 was considered statistically significant at the 95% confidence interval. Ethical clearance for the study was obtained from the Ethical Committee of Chittagong Medical College. Study details were explained to participants in Bangla, and for those unable to understand Bangla, the information was translated into local languages. Written informed consent was obtained from all participants prior to enrollment. For children aged 12–18 years, both guardian consent and patient assent were obtained. In the case of unconscious patients, consent was taken from their attending adult guardians. As this was a purely observational study, no experimental intervention was performed. Patients who did not consent received standard treatment as per ward protocol. Participants were free to withdraw consent at any stage without any effect on their medical management. No additional or invasive investigations were conducted beyond routine clinical requirements.

RESULTS

38 cases of definite Pyrethroid Poisoning were included in the study. During observation, 3 patients dropped out of the total of 38 patients. Therefore, daily follow-up and investigations were carried out on the remaining 35 patients. A total of 761 patients were registered as agrochemical poisoning from May 2017 to April 2018. Out of these, only in 222 cases was sample identification possible by studying the types and chemical composition of poisons. Out of 222 cases, only 38 cases were confirmed as Pyrethroids according to the case definition.

Table I shows that the majority of the patients (39.47%) were of 12-19 years of age. Mean age in case of male (25.33±10.34) years and in case of female (25.36±12.02) years. The majority of the cases were male (63%). [Table I]

Table – I: Age and Sex Distribution of the Pyrethroid Poisoning Patients

Age Group	Male	Female	Total
12-19 Years	9	6	15
20-29 Years	8	4	12
30-39 Years	5	3	8
>40 Years	2	1	3
Total	24	14	38

Table II shows that the majority of cases were farmers (29%), followed by students (29%). [Table II]

Table – II: Occupation of the Pyrethroid poisoning patients

Occupation	Frequency	Percentage
Farmer	11	29.00%
Businessman	3	8.00%
Housewife	7	18.21%
Student	11	29.00%
Worker	4	10.53%
Service Holder	1	2.63%
Unemployed	1	2.63%
Total	38	100.00%

Table III shows that the majority of the cases were married (68.4%) [Table III]

Table – III: Marital status of the Pyrethroid patients

Marital Status	Frequency		Total	Percent
	Male	Female		
Married	15	11	26	68.4
Unmarried	09	03	12	31.6
Total	24	14	38	100.0

ISSN: 2663-9491 e-ISSN: 2789-6897

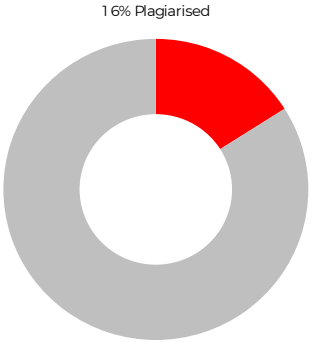

Figure – 1: Monthly Distribution of Pyrethroid Poisoning Cases

Figure 1 shows that the majority of the patients came in January (21.05%), then July (13.15%), and December (13.15%) [Figure 1]

There is a significant difference in time difference between ingestion of poison and admission in CMCH between the patients from Chattogram City and patients outside of Chattogram district (p= 0.006 at 95% CI). This suggests a significant delay in the initiation of treatment among these two groups of patients. Though there was no significant difference observed between patients of Chattogram district and patients out of Chattogram district. [Table IV]

Table – IV: Mean period of interval between ingestion of poison and initiation of treatment

Area of The Patient	Difference between time of Ingestion of poison and Initiation of Treatment (Mean ± SD) Hours	Comparison group	P value*
Chattogram City	3.5± 2.83	Chattogram District	0.130
		Out of Chattogram District	0.006
Chattogram District	6.33± 2.22	Chattogram City	0.130
		Out of Chattogram District	0.184
Out of Chattogram District (Cox's Bazar, Feni)	8.9± 5.78	Chattogram City	0.006
		Chattogram District	0.184
Overall	6.26 ± 4.03 Hours		

**p value calculated by One-way ANOVA (Post Hoc test).*

Table V shows that the majority of the pyrethroids (52.63%) were cypermethrin. Lambda-cyhalothrins were 39.47% and Fenvalerates were 7.90%. All three types were of type II Pyrethroids. In the case of Cybromethrin, the most common compounds were Ustaad and Leader. In the case of Lambda-cyhalothrins, the most common compounds were Fighter and Effect. [Table V]

Table – V: Types of pyrethroids

Cypermethrins		Lambda- Cyhalothrins		Fenvalerates	
Chemical Names	No	Chemical names	No	Chemical names	No
Ustaad	4	Fighter	4	Valafen	2
Leader	3	Effect	4	Milfen	1
Superthrin	2	Oxit	2		
Sumnethrin	2	Cyclone EC	2		
Limper	2	Karate	2		
Howthrin	2	Boxer	1		
Cyrex	2				
Fencord	1				
Ripcord	2				
Total	20	Total	15	Total	3

Table VI showing 78.94% of the patients were treated initially with atropine and 36.84% were with praildoxime. 86.84% of the patients were with PPI (Omeprazole/Pantoprazole) and 42.10% were given broad spectrum antibiotics. [Table VI]

Table – VI: Medications given to the Pyrethroid patients

Name of Drug	Drugs Given (No./Percentage)	
	Yes	No
Atropine	30 (78.94%)	08(21.06%)
Praildoxime	14 (36.84%)	24 (63.16%)
Antibiotics (i.e. Ceftriaxone)	16 (42.10%)	22 (57.89%)
PPI (i.e. Omeprazole)	33 (86.84%)	05 (13.16%)

ISSN: 2663-9491 e-ISSN: 2789-6897

Table VII shows, on admission majority of the patients reported nausea (100%) followed by blurred vision (94.73%), increased salivation (92.10%), dizziness (89.47%) and psychiatric symptoms (hallucination, restlessness) (15.80%). [Table VII]

Table – VII: Symptoms of the Pyrethroid patients on admission (*n*=38)

Clinical features	Present
Dizziness	34(89.47%)
Nausea	38 (100%)
Blurred Vision	36(94.73%)
Increased Salivation	35(92.10%)
Diaphoresis	06(15.80%)
Headache	04(10.52%)
Psychiatric Symptoms (i.e. Hallucination, Restlessness)	06(15.80%)

Table VIII shows that the majority of the patients showed increased salivation (42.86%), abdominal pain (25.72%), loose motion (8.6%), and dysuria (14.30%). [Table VIII]

Table – VIII: Symptoms on 2nd day of poisoning (48 hours) (*n*=35)

Clinical features	Present
Abdominal pain	09(25.72%)
Increased Salivation	15(42.86%)
Loose motion	03(8.60%)
Dysuria	05(14.30%)
Instability to walk	07(20%)

Table IX showing that the most common sign elicited was constricted pupil (68.40%) (All the patients were treated with Atropine initially). Second common sign was raised temperature/ fever (52.63%). Out of 20 patients Atropine was given to 15 patients initially. 6 patients developed instability during walking, of them only 2 were treated with Atropine. 7 patients developed tachycardia and restlessness, out of them, 5 patients were treated with Atropine initially. Only one patient developed respiratory distressed and was transferred to Intensive care unit. [Table IX]

Table – IX: Signs of Pyrethroid Poisoning on Admission (*n*=35)

Clinical Features	Present
Constricted pupil	26(68.40%)
Fever	20(52.63%)
Instability/ataxia	06(15.80%)
Tachycardia	07 (18.40%)
Restlessness	07(18.40%)
Acute confusional state(GCS<13)	03(7.90%)
Respiratory Distress	01(2.63%)

Table X showing majority of the patients had dilated pupil (28.57%) and all of them were treated with Atropine initially. Fever is an important sign of Pyrethroid poisoning as these 5 patients were not treated with Atropine. Jaundice was present on 4 patients and out of them one patient was suffering from chronic liver diseases. Rest of the 3 patients had no history of prior liver diseases. One patient present focal neurological sign as he developed Ischemic stroke. [Table X]

Table – X: Signs on 2nd day of poisoning (48 hours) (*n*=35)

Clinical features	Present
Dilated pupil	10(28.57%)
Fever	05(14.28%)
Instability/ataxia	04(11.42%)
Reduced muscle tone	02 (5.72%)
Jaundice	04(11.42%)
Tenderness of abdomen	09(25.72%)
Focal neurological signs (stroke)	01(2.85%)

Table XI showing haematological and biochemical changes in Pyrethroid poisoning patients. [Table XI]

Table – XI: Investigation findings (Hematological and Biochemical) of the Pyrethroid patients (*n*=35)

Name of Investigation	Mean ± SD	Range
Hb(gm/dl)	12.35 ± 1.39	9.8-14.2
Neutrophil Count in Percentage	74.30 ± 9.63	50-85
Lymphocyte Count in Percentage	22.40 ± 9.14	10-36
No. of Pus Cells in Urine/HPF	5.18 ± 8.31	1-25
Level of Blood Glucose (mmol/L)	4.95 ± 2.52	4.4-7.4
Serum Creatinine Level (mg/dl)	0.97 ± 0.23	0.5-1.8
ALT Level (IU/L)	39.96 ± 20.89	16-244
Serum Na Level (mmol/L)	139.90 ± 2.88	135-145
Serum K Level (mmol/L)	4.23 ± 0.78	3.1-5.4
Serum Cl Level (mmol/L)	101.41 ± 3.56	93-105
Serum HCO3 Level (mmol/L)	24.52 ± 0.75	24-25

Table XII showing Neutrophilic leukocytosis in 13 patients, raised ALT in 06 (one patient was suffering from Chronic liver disease and rest 5 had no previous history of liver diseases). Pus cells and RBC in urine in 08 patients (all of them were catheterized). Mild hyperglycemia in 05 patients (one patient was diabetic and other 4 patients were non-diabetic). Raised S. Creatinine was present in 3 patients (one patient was suffering from diarrhea and hypotension). [Table XII]

Table – XII: Abnormal laboratory findings in Pyrethroid Poisoning (*n*=35)

Abnormal findings	Present
Hyperglycemia	05(14.28%)
Neutrophilic leucocytosis	13(37.14%)
Raised ALT level	06(17.14%)
Pus cells in urine	08(22.85%)
RBC in urine	08(22.85%)
Raised serum creatinine	03(8.57%)

Table XIII shows that majority of the patients (94.28%) were improved, whereas 5.72% died. Out of two dead patients, one male (65 years of age) died due to Ischemic stroke followed by cardiac arrest (had a previous history of Hypertension and Diabetes). Another patient, a female (36 years of age), died due to Aspiration pneumonia and respiratory failure. The deaths of the two cases were related to Pyrethroid poisoning, as in both cases, large doses of poisons were ingested and there was a history of delayed arrival at the hospital. [Table XIII]

ISSN: 2663-9491 e-ISSN: 2789-6897

Table – XIII: Clinical outcome of pyrethroid Poisoning (*n*=35)

Clinical Outcome	Yes			No		
	Male	Female	Total	Male	Female	Total
Improved	22	11	33 (94.28%)	01	01	02 (05.72%)
Died (stroke, Respiratory failure)	01	01	02 (05.72%)	22	11	33(94.28%)

DISCUSSION

The present descriptive observational study evaluated the epidemiological, clinical, and laboratory profiles, along with the outcomes, of pyrethroid poisoning cases admitted to Chattogram Medical College Hospital. Although the estimated sample size was 196, a total of 38 definite pyrethroid poisoning cases were included within the study period, of which 35 patients completed full follow-up. Out of 761 agrochemical poisoning cases recorded from May 2017 to April 2018, only 222 cases had identifiable chemical samples, and 38 (4.9%) were confirmed as pyrethroid poisoning. This indicates a higher incidence compared to earlier reports from Bangladesh, where the prevalence was reported as only 0.5% by Bari et al.[3] Based on a multicenter analysis, Amin et al. predicted an incidence of 2.5% [8]. The increased rate in the present study may be due to active efforts to identify the poison container and the recent shift toward non-organophosphorus compounds in agriculture. In this study, males were more affected than females (male: female = 1.7:1), which is consistent with previous findings by Haque et al. and Jesslin et al., who reported male predominance in pesticide poisoning [4,6]. Men are often more exposed to agricultural chemicals and have easier access to toxic agents, explaining this gender distribution. According to the World Health Organization (WHO) report, men die by suicide nearly three times more often than women in high-income countries, while the rates are more equal in low- and middle-income nations [5]. In Bangladesh, where pesticide ingestion remains a common method of deliberate self-harm, this pattern aligns with global data. The age distribution revealed that most victims were adolescents and young adults, particularly those aged 12–19 years (39.5%) and 20–29 years (31.6%). Similar findings were reported by Chowdhury et al., who noted that individuals under 30 years are most vulnerable to self-poisoning [7]. Younger people, especially students, often experience emotional instability and academic or social stress that predisposes them to impulsive acts. Farmers comprised another major group (29%), reflecting occupational exposure and easy pesticide availability, similar to the observations of Gunnel et al. and Chowdhury et al.[5, 7] A clear seasonal trend was noted, with higher incidence during December–January and July–August, which correspond to the main crop cultivation periods in Bangladesh. Increased pesticide availability during these agricultural seasons likely raises both accidental and intentional ingestion. The time interval between ingestion and initiation of treatment significantly varied by residence. Patients from Chattogram city arrived within 3.5 ± 2.8 hours, whereas those from outside the district required 8.9 ± 5.8 hours ($p = 0.006$). This delay in rural patients is critical, as early hospital presentation is a key determinant of survival. Chemical identification showed three type II pyrethroids—cypermethrin (52.63%), lambda-cyhalothrin (39.47%), and fenvalerate (7.9%). Similar findings were reported by He et al. and Yang et al., who documented that these compounds are among the most frequent pyrethroid agents in poisoning cases [9,13]. In the present study, the common trade names were *Ustaad* and *Leader* (cypermethrin-based) and *Fighter* and *Effect* (lambda-cyhalothrin-based), reflecting their wide commercial use in Bangladesh. Clinically, almost all patients presented with nausea (100%), blurred vision (94.7%), increased salivation (92.1%), and dizziness (89.5%). These features are comparable to those described by He et al., who reported nausea, vomiting, dizziness, and abdominal pain as predominant symptoms [9,10]. Psychiatric manifestations such as restlessness and hallucinations occurred in 15.8% of cases, possibly related to both atropine toxicity and pyrethroid neurotoxicity. Instability while walking and ataxia, seen in 20% of patients, were recognized as features of moderate poisoning in the study by Bradberry et al.[14] Many patients in this series were initially misdiagnosed as organophosphorus (OPC) poisoning and treated with atropine and pralidoxime before confirmation. Although atropine may reduce secretions, excessive or unnecessary use can cause blurred vision, tachycardia, and dilated pupils, confounding the clinical picture. Similar diagnostic confusion was noted by Chowdhury et al. and Proudfoot et al., highlighting the importance of early toxicological confirmation and the need to differentiate pyrethroid poisoning from OPC poisoning based on clinical and contextual cues [7, 15]. Laboratory abnormalities in this study included neutrophilic leukocytosis (37.1%), hyperglycemia (14.3%), elevated ALT (17.1%), and raised serum creatinine (8.6%). Comparable findings were reported by Yang et al. [13] and Bradberry et al., who noted leukocytosis, mild hepatic enzyme elevation, and transient renal dysfunction in acute pyrethroid poisoning [14]. These alterations are likely due to oxidative stress, inflammatory activation, and cellular toxicity, as described by Ray and Forshaw and Gargouri et al.[15, 16] Hyperglycemia observed in the present study may be explained by catecholamine surge and altered carbohydrate metabolism following exposure, a mechanism demonstrated in animal studies by Abdel-Khalik et al.[17] Regarding outcomes, 94.3% of patients recovered completely, while two deaths (5.7%) occurred. One 65-year-old hypertensive and diabetic male died from ischemic stroke and cardiac arrest, and a 36-year-old female died due to aspiration pneumonia and respiratory failure. Similar rare fatal outcomes were reported by Yang et al. [13] and Bradberry et al., usually resulting from respiratory failure or aspiration rather than direct pyrethroid toxicity [14]. Both fatal cases in this study involved delayed hospital admission and the presence of comorbidities, emphasizing the role of early treatment and supportive care in survival. Overall, the prognosis of pyrethroid poisoning is favorable when appropriate supportive management is instituted. However, misdiagnosis as OPC poisoning and injudicious use of atropine can complicate recovery. He et al. [9] and Ray et al. [16] emphasized that there is no specific antidote for pyrethroid poisoning, and therapy should focus on symptomatic management, respiratory support, and prevention of complications.

Limitation of the Study:

This study was limited by being single-center, having a small sample size, and not analyzing the underlying causes of observed complications.

CONCLUSION

A hospital-based clinical study was conducted on 38 patients. The most affected age group was 12–19 years, followed by 20–29 years. The majority of the poisoning cases occurred among farmers and students. There was a significant difference in the time interval between patients coming from inside the city and

ISSN: 2663-9491 e-ISSN: 2789-6897

those coming from outside the city. Pyrethroid ingestion caused immediate nausea and vomiting, followed by increased salivation and secretions. After 4–8 hours, patients developed dizziness, headache, blurred vision, palpitations, and instability. Some patients experienced an acute confusional state, restlessness, fever, mild jaundice, and dysuria. Most symptoms persisted for 24–48 hours, and the patients' condition recovered spontaneously within 5–6 days. Some hematological and biochemical changes were also observed, including neutrophilic leukocytosis, mild hyperglycemia, raised ALT, and elevated creatinine levels. Death due to poisoning occurred very rarely and was associated with comorbidities such as stroke and respiratory failure.

RECOMMENDATIONS

- Collection of poison bottles is crucial in case of all types of poisoning.
- Poisoning studies of Bangladesh require stricter criteria for case definition.
- All doctors should have the OPC and Non-OPC poison lists for quick identification.
- A guideline protocol should be published for specific treatment.
- A large-scale prospective study by application of sample identification to determine the nature of agrochemical poisons will increase the accuracy of the findings.

Funding: *No funding sources*
Conflict of interest: *None declared*

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