Management of organophosphorus pesticide poisoning in Assiut University Children's Hospital: A clinical audit

Azza A. Eltayyeb, Eman Fathala Gad, Asmaa Nagah Abd Elaal

Department of Pediatrics, Faculty of Medicine, Assiut University, Assiut, Egypt

Correspondence to Dr. Asmaa Nagah Abd Elaal, BSC of Medicine and Surgery, Department of Pediatric , Faculty of Medicine, Assiut University, Assiut, Egypt.
Postal code: 71717;
Tel: +20 1062974738;
e-mail: Asmaanagah1990@gmail.com

Received 15 December 2019
Revised 18 December 2019
Accepted 20 January 2020
Published 11 June 2021

Journal of Current Medical Research and Practice
2021, 6:174–180

Background
Acute organophosphorus (OP) pesticide self-poisoning is a major global problem. Although pesticide-poisoned patients make high demands on intensive care facilities in industrialized countries, it is in the developing world that practically all deaths occur.

Objective
The aim of this clinical audit is to study to how extent we are following the guidelines of management of OP poisoning in the emergency unit, intermediate care unit, and ICU at Assiut University Children's Hospital.

Patients and methods
The present study included 40 children presented with OP poisoning and admitted to the ICU and intermediate care unit of Assiut University Children's Hospital over a period of 6 months from the October 1, 2018 to March 31, 2019.

Results
The study included 40 patients with OP poisoning. Their ages ranged from 1 to 18 years, the most commonly presented age group was from 1 to 5 years. Ingestion was the most common type of exposure; accidental exposure was more common than self-harm; vomiting and diarrhea was the most common presentation; plasma cholinesterase enzyme was not done in 90% of cases; decontamination and atropinization were done in most of the cases. The most common complication was shock. Regarding the outcome of cases, 90% of the studied cases improved and 10% of them died.

Conclusion
International guidelines for the management of OP poisoning had been followed by personnel working at the emergency unit, intermediate care unit, and ICU at Assiut University Children's Hospital regarding treatment lines and that some of the defaults are due to poor resources and lack of medication.

Keywords:
Assiut University Children's Hospital, audit, organophosphorus poisoning

J Curr Med Res Pract 6:174–180

2357-0121

Introduction

Acute organophosphorus (OP) pesticide poisoning is an increasing worldwide problem, particularly in rural areas. Intentional ingestion of OP is associated with a high mortality rate [1,2]. The WHO estimates the incidence of pesticide poisoning to be three million cases per year, leading to more than 250 000 deaths annually [3].

Management of OP poisoning has two components: general management which includes immediate assessment and management of disturbances in the airway, breathing, and circulation and decontamination (skin and gastric). Further steps are based on risk assessment and observations during continuous monitoring [4]. Antidotes are muscarinic antagonists (usually atropine), oximes (usually pralidoxime), and benzodiazepines [5].

Aim

The clinical audit study aims to assess the adapted protocols of diagnosis and management of OP poisoning in Assiut University Children's Hospital.

Patients and methods

Study site: emergency unit, intermediate care unit and PICU at Assiut University Children's Hospital. Informed consent was obtained from every patient 's parents according to the guidelines of the Committee of Medical Ethics of Assiut University Hospitals. The study was approved and monitored by the Medical

Ethics Committee, Assiut Faculty of Medicine. IRBno:17100854.

Type of the study: audit study (descriptive).

Duration of study: 6 months.

Study population: the target population of this study included all patients diagnosed as OP poisoning and admitted to Assiut University Children's Hospital.

Inclusion criteria

(1) The study included all children with OP poisoning admitted to Assiut University Children's Hospital. The age of the patients ranged from 1 to 18 years.
(2) Diagnosis was based on a history of exposure, the characteristic clinical signs, smell of pesticides or solvents, and reduced butyrylcholinesterase or acetylcholinesterase activity in the blood.

Exclusion criteria

Patients exposed to other intoxication or poisoning as drugs or scorpion sting or other agents.

Methods

The study was approved and monitored by the Medical Ethics Committee, Assiut Faculty of Medicine (IRB no.: 17100854).

Cases of OP poisoning were diagnosed according to clinical suspicion from history, physical examination, and laboratory investigation.

Approach to clinical diagnosis of a case of OP poisoning according to the guidelines regarding:

(1) Full history taking: name, age, sex, occupation, and residence.
(2) History included: present history, exposure (ingestion,dermal exposure,inhalation),therapeutic history, and history of decontamination.
(3) Examinations included: general examination, vital signs, abdomen, chest, cardiac and neurological examination.
(4) Clinical manifestations for signs of poisoning: such as special odor, miosis, signs of increased secretion, sweating, vomiting, shock, pulmonary edema, and urine output.
(5) Laboratory investigations: such as serum electrolytes, serum urea, serum creatinine, complete blood count, creatine phosphokinase, blood glucose level, arterial blood gases, and plasma cholinesterase.
(6) Imaging studies: chest radiograph and ECG.
(7) Specific investigation according to the manifestation when needed.
(8) Management of OP poisoning is done by contamination, stabilization of the airway, stabilization of circulatory support, or by using antidote therapy such as atropine, pralidoxime or benzodiazepines and mechanical ventilation if needed.

Results

The study included 40 patients with OP poisoning. Their ages ranged from 1 to 18 years, 20 cases were men and 20 cases were women .

All cases (100%) were asked about the type of exposure; 38 (95%) cases were exposed by the ingestion route and two (5%) cases were exposed by the inhalation route. (Fig. 1).

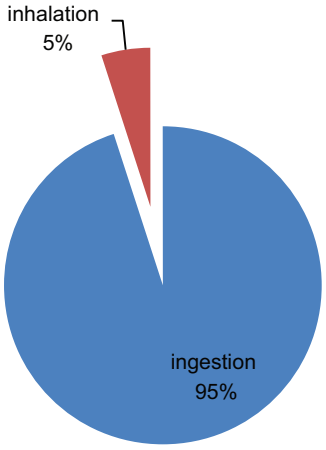
According to the guidelines, the following symptoms were asked about: abdominal cramp in 23 (57.5%) cases, anorexia in 24 (60%) cases, lacrimation in 32 (80%) cases, salivation in 32 (80%) cases, vomiting in 40 (100%) cases, diarrhea in 33 (82.5%) cases, dyspnea in 32 (80%) cases, cough in 28 (70%) cases, incontinence in 29 (72.5%) cases, involuntary defecation in 32 (80%) cases, confusion in 36 (90%) cases, and convulsion in 34 (85%) cases. Clinical presentations of the cases were: abdominal cramp in 15 (65.2%) cases, anorexia in 16 (66.7%) cases, lacrimation in 20 (62.5%) cases, salivation in 22 (68.8%) cases, vomiting in 30 (75%) cases, diarrhea in 24 (72.7%) cases, dyspnea in 18 (56.2%) cases, cough in 17 (60.7%) cases, incontinence in 14 (48.3%) cases, involuntary defecation in nine (28.1%) cases, confusion in 29 (80.6%) cases, and convulsion in 11 (32.4%) cases (Fig 2).

Results

The study was conducted at the emergency unit, intermediate care unit, and ICU at Assiut University Children's Hospital and aimed to observe health-care provider's performance after they get trained on guidelines for the management of OP poisoning[6] from October 1, 2018 to March 30, 2019. It was conducted using checklist tables including all data that was designed according to the guidelines. The residents were educated on how to follow the guidelines at the start of the study and they were evaluated for each step.

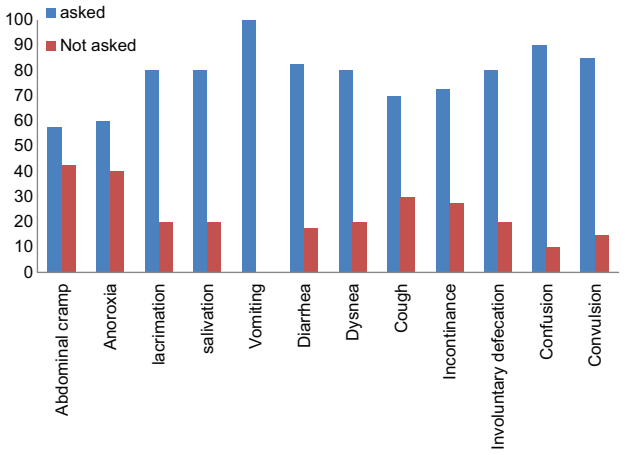
A good point noticed during observation is that all cases in the emergency unit, intermediate care unit, and ICU are reviewed by assistant lecturers and this

Figure 1



Recorded data about the type of exposure among cases with organophosphorus poisoning.

Figure 2



Clinical symptoms among cases with organophosphorus poisoning.

may explain the significantly high compliance to the standards which was reassuring and satisfying.

Our study, according to the guidelines, provides important data to be fulfilled by the history and examination to improve the management of OP poisoning.

Recorded data about history:

(1) Data of age, sex were recorded in 100% of cases. It was more common in the age group from 1 to 5 years by 27 (67.5%) cases, group from 5 to 10 years by five (12.5%) cases, and a group of more than 10 years by eight (20%) cases. This study showed that the most common age presentation was from 1 to 5 years. There was no significant difference in sex distribution. Twenty (50%) cases were men and 20 (50%) cases were women. Data about residence and place of exposure were done in 80% of cases. Twenty-one (52.5%) cases were from rural areas and 11 (27.5%) cases were from urban areas. Twenty-six (65%) cases were exposed to poison indoor and six (15%) cases were exposed outdoor (Table 1).

Recorded data about general examination:

(1) According to the guidelines, the following signs were examined: level of consciousness in 40 (100%) cases, pupil examination in 39 (97.5) cases, pallor in 34 (85%) cases, cyanosis in 37 (92.5%) cases, sweating in 35 (87.5%) cases, rhinorrhea in 31 (77.5%) cases, and special odor in 24 (60%) cases. Clinical presentation of the cases were: disturbed conscious level in 23 (57.5%) cases, pinpoint pupil in 32 (82.05) cases, pallor in 14 (41.2%) cases, cyanosis in 11 (29.7%) cases, sweating in 25 (71.4%) cases, rhinorrhea in 19 (61.3%) cases, and characteristic odor in six (25%) cases.

Table 1 Demographic data of the cases with organophosphorus poisoning (*n*=40)

	n (%)
Age groups (years)	
1-5	27 (67.5)
5-10	5 (12.5)
>10	8 (20)
Mean±SD	5.9±4.64
Sex distribution	
Boys	20 (50)
Girls	20 (50)
Residence	
Rural	21 (52.5)
Urban	11 (27.5)
Place of exposure	
Indoor	26 (65)
Outdoor	6 (15)

Temperature was recorded in 77.5% (48.5% were normal, 41.9% were hypothermia, and 9.7% were hyperthermia). Blood pressure was recorded in 97.5% of cases (58.97% were normal, 41.03% were hypotensive, and 3% were hypertensive). Heart rate was recorded in 95% of cases (52.6% of cases were normal, 15.8% were bradycardic, and 31.6% of cases were tachycardic). Respiratory rate was recorded in 97.5% of cases (33.33% of cases were normal, 17.95% were bradypnea, and 48.72% of cases were tachypnea) (Table 2).

Recorded data about systemic examination (Table 3):

(1) Clinical examination of respiratory system retraction was examined in 35 (85%) cases [30 (88.2%) cases had retraction]. Grunting was examined in 32 (80%) cases [19 (59.4%) cases were presented with grunting]. Chest auscultation was done in 100% of cases [90% of cases had equal air entry, wheezing was present

Table 2 General examination among cases with organophosphorus poisoning (*n*=40)

	Done [*n* (%)]	Not done [*n* (%)]
Level of conscious:	40 (100)	0
Conscious	17 (42.5)	
Disturbed	23 (57.5)	
Pupil examination	39 (97.5)	1 (2.5)
Normal	7 (17.95)	
Pinpoint pupil	32 (82.05)	
Pallor	34 (85)	6 (15)
Present	14 (41.2)	
Not present	20 (58.8)	
Cyanosis	37 (92.5)	3 (7.5)
Present	11 (29.7)	
Not present	26 (70.3)	
Sign of intoxication		
Sweating	35 (87.5)	5 (12.5)
Present	25 (71.4)	
Not present	10 (28.6)	
Rhinorrhea	31 (77.5)	9 (22.5)
Present	19 (61.3)	
Not present	12 (38.7)	
Special odor	24 (60)	16 (40)
Present	6 (25)	
Not present	18 (75)	

in 13 (32.5%) cases, crepitation was present in 33 (82.5%) cases, and pulmonary edema was present in three (7.5%) cases].

(2) Regarding cardiac examination signs of heart failure were examined in 62.5% of cases. Signs of arrhythmia were examined in 38 (95%) cases [52.6% of cases had normal rhythm and 47.4% of cases had abnormal rhythm]. Cardiac examination was done for other signs in 26 (65%) cases.

(3) Neurological examination in which twitches of the facial muscle was examined in 32 (80%) cases [20 (62.5%) cases had twitches in their faces]. Tremor was examined in 26 (65%) cases [12 (46.2%) cases presented with tremor]. Gait was examined in 18 (45%) cases [17 (94.4%) cases had a normal gait, one (5.6%) case had abnormal gait]. Muscle tone was examined in 39 (97.5%) cases [20 (53.3%) cases had normal tone, 19 (48.7%) cases had hypotonia]. Reflexes were examined in 36 (90%) cases [22 (61.1%) cases had normal reflexes, 14 (38.9%) cases had hyporeflexia]. Cranial nerves were examined in 19 (47.5%) cases, all of them were found to be intact. Hallucination was examined in 25 (62.5%) cases [three (12%) cases were presented with hallucination]. Abnormal movement was examined in 24 (60%) cases [three (12.5%) cases had abnormal movement].

(4) Urine output was recorded in 18 (45%) cases [13 (72.2%) cases had normal urine output, five (27.8%) cases had abnormal urine output].

Table 3 Systemic examination of the studied children (*n*=40)

	Done [*n* (%)]	Not done [*n* (%)]
Respiratory system		
Retraction	34 (85.0)	6 (15.0)
Present	30 (88.2)	
Not present	4 (11.8)	
Grunting	32 (80.0)	8 (20.0)
Present	19 (59.4)	
Not present	13 (40.6)	
Chest auscultation	40 (100)	0 (0)
Equal air entry	36 (90.0)	
Wheeze	13 (32.5)	
Crepitation	33 (82.5)	
Pulmonary edema	3 (7.5)	
Cardiac examination		
Signs of heart failure	25 (62.5)	15 (37.5)
Present	0	
Not present	25 (100)	
Arrhythmia	38 (95)	2 (5)
Normal rhythm	20 (52.6)	
Abnormal rhythm	18 (47.4)	
Other signs	26 (65)	14 (35)
Present	0	
Not present	26 (100)	
Neurological examination		
Twitches of facial muscle	32 (80)	8 (20)
Present	20 (62.5)	
Not present	12 (37.5)	
Tremor	26 (65.0)	14 (35.0)
Present	12 (46.2)	
Not present	14 (53.8)	
Gait	18 (45.0)	22 (55.0)
Normal	17 (94.4)	
Abnormal	1 (5.6)	
Muscle tone	39 (97.5)	1 (2.5)
Hypotonia	19 (48.7)	
Normal	20 (51.3)	
Reflexes	36 (90)	4 (10)
Normal	22 (61.1)	
Hyporeflexia	14 (38.9)	
Cranial nerves	19 (47.5)	21 (52.5)
Intact	19 (100)	
Hallucination	25 (62.5)	15 (37.5)
Present	3 (12)	
Not present	22 (88)	
Abnormal movement	24 (60)	16 (40)
Present	3 (12.5)	
Not present	21 (87.5)	
Urine output	18 (45)	22 (55)
Normal	13 (72.2)	
Abnormal	5 (27.8)	
Abdominal examination		
Intestinal sound	38 (95)	2 (5)
Normal	33 (86.8)	
Absent	5 (13.2)	
Abdominal distention	37 (92.5)	3 (7.5)
Present	7 (18.9)	

(5) Abdominal examination of intestinal sound was examined in 38 (95%) cases [33 (86.8%) cases had normal intestinal sound, five (13.2%) cases had

absent intestinal sound]. Abdominal distention was examined in 37 (92.5%) cases which was present in seven (18.9%) cases.

Recorded data about laboratory investigations (Table 4):

The diagnosis was confirmed by measuring plasma cholinesterase levels. Low plasma ChE levels support the diagnosis of OP poisoning [7].

(1) Random blood glucose was done in 30 (75%) cases [18 (60%) cases were normal, nine (30%) cases were hyperglycemic, three (10%) cases were hypoglycemic]. White blood cell count was done in 33 (82.5%) cases [18 (54.5%) cases had normal count and 15 (45.5) cases had leukocytosis]. Hemoglobin level was done in 33 (82.5%) cases [24 (72.7%) cases were normal and nine (27.3) cases had low hemoglobin levels].
(2) Platelet count was done in 33 (82.5%) cases [29 (87.9%) cases had normal count and four (12.1) cases had thrombocytosis].
(3) Serum electrolyte and kidney function tests were done in 33 (82.5%) cases which in serum creatinine examination [32 (96.97%) cases had normal result, one (3.03) case had increased level]. BUN examination [32 (96.97%) cases had normal result, one (3.03) case had increased level]. Serum sodium examination [31 (93.94%) cases were normal and two (6.06) cases were hypernatremic]. Serum potassium examination [33 (100%) cases were normal].
(4) Plasma cholinesterase enzyme was done in four (10%) cases and all of them showed decreased levels.
(5) ECG was done in 18 (45%) cases [12 (66.7%) cases had normal findings, six (33.3%) cases had abnormal finding]. Chest radiograph was done in 30 (75%) cases [25 (83.3%) cases had normal findings and five (16.7%) cases had abnormal findings]. Arterial blood gases was done in 26 (65%) cases of which 13 (50%) cases were normal.

Recorded data about management (Table 5):

(1) The present study showed that decontamination was done in 35 (87.5%) cases. The secure airway was done in 100% of cases. Circulatory support was done in 55% of cases. Atropine was given in adjusted doses in 100% of cases with frequency of repetition [once in 12 (30%) cases, twice 13 (32.5%) cases, three times in six (15%) cases, four times in five (12.5%) cases, five times in three (7.5%) cases and 6 times in one (2.5%) case].

Table 4 Recorded data about laboratory and imaging study of cases with organophosphorus poisoning (*n*=40)

Investigation	Done [*n* (%)]	Not done [*n* (%)]
Random blood glucose	30 (75)	10 (25)
Normal	18 (60)	
Increased	9 (30)	
Decreased	3 (10)	
WBCs	33 (82.5)	7 (17.5)
Normal	18 (54.5)	
Abnormal (increased)	15 (45.5)	
Hemoglobin level	33 (82.5)	7 (17.5)
Normal	24 (72.7)	
Abnormal (decreased)	9 (27.3)	
Platelet count	33 (82.5)	7 (17.5)
Normal	29 (87.9)	
Abnormal (increased)	4 (12.1)	
Serum sodium	33 (82.5)	7 (17.5)
Normal	31 (93.94)	
Abnormal (increased)	2 (6.06)	
Serum potassium	33 (82.5)	7 (17.5)
Normal	33 (100)	
Abnormal	0	
Blood urea nitrogen	33 (82.5)	7 (17.5)
Normal	32 (96.97)	
Abnormal (increased)	1 (3.03)	
Serum creatinine	33 (82.5)	7 (17.5)
Normal	32 (96.97)	
Abnormal (increased)	1 (3.03)	
Plasma cholinesterase enzyme	4 (10)	36 (90)
Decreased	4 (10)	
ECG	18 (45)	22 (55)
Normal	12 (66.7)	
Abnormal	6 (33.3)	
Chest radiograph	30 (75)	10 (25)
Normal	25 (83.3)	
Abnormal	5 (16.7)	
Arterial blood gases	26 (65)	14 (35)
Normal	13 (50)	
Abnormal	13 (50)	

WBC, white blood cell.

(2) Benzodiazepines were given in an adjusted dose in 22.5% of cases which was needed. Mechanical ventilation was needed in 15% of cases. Anticonvulsive drugs were needed in 22.5% of cases. Inotropic drugs were needed in 22.5% of cases. Cardioversion was needed in 12.5% of cases. Diuretic was needed in 2.5% of cases.

Recorded data about outcome and complications (Tables 6 and 7):

(1) In this study, the detected complications were: coma was present in nine cases (22.5%), seizure was present in 11 (27.5%) cases, respiratory failure was present in six (15%) cases, respiratory acidosis was present in 13 (32.5%) cases, cardiogenic shock was present in four (10%) cases, and hypovolemic shock was present in 13 (32.5%) cases.

Table 5 Recorded data about the management of cases with organophosphorus poisoning (*n*=40)

Treatment	Done [*n* (%)]	Not done [*n* (%)]
Decontamination	35 (87.5)	5 (12.5)
Airway support	40 (100.0)	0
Circulatory support	22 (55)	18 (45)
Atropinization	40 (100.0)	0
Once	12 (30.0)	
Twice	13 (32.5)	
Three times	6 (15)	
Four times	5 (12.5)	
Five times	3 (7.5)	
Six times	1 (2.5)	
Pralidoxime	0	40 (100.0)
Need of benzodiazepines	9 (22.5)	31 (77.5)
Need for mechanical ventilation	6 (15)	34 (85)
Need of anticonvulsants	9 (22.5)	31 (77.5)
Need of inotropic drugs	9 (22.5)	31 (77.5)
Need of cardioversion	5 (12.5)	35 (87.5)
Need of diuretics	1 (2.5)	39 (97.5)

Table 6 Complications of organophosphorus poisoning in the studied children (*n*=40)

Complication	*n* (%)
Coma	9 (22.5)
Seizure	11 (27.5)
Respiratory failure	6 (15)
Respiratory acidosis	13 (32.5)
Shock	17 (42.5)
Hypovolemic shock	13 (32.5)
Cardiogenic shock	4 (10)

Table 7 Outcome organophosphorus poisoning in the studied children (*n*=40)

	n (%)
Outcome	
Improved	36 (90.0)
Died	4 (10.0)

(2) The outcome of the studied cases with OP poisoning showed that four (10%) cases died and 36 (90%) cases improved.

Conclusion

International guidelines for the management of OP poisoning had been followed by personnel working at the emergency unit, intermediate care unit, and ICU at Assiut University Children's Hospital regarding treatment lines and that some of the default is due to poor resources and lack of medication.

Recommendations

(1) Applying the standard guidelines in health-care centers about the early referral of children with OP poisoning to higher levels and the importance of the stabilization of the patient before and during transportation.
(2) Awareness among physicians at primary, secondary, and tertiary level centers regarding decontamination and its importance in decreasing absorption of the poison.
(3) Awareness among physicians regarding the importance of gastric lavage is indicated once the patient is stabilized. It is recommended to be carried out within 1–2 h of ingestion and repeated after 2–3 h.
(4) Time interval between the ingestion of poison and arrival to health care should be recorded.
(5) A careful history should be taken regarding abdominal cramp, anorexia, cough, and incontinence.
(6) Careful examination should be done regarding special odor, temperature, signs of heart failure, other cardiac signs, tremors, cranial nerves, and abnormal movement.
(7) Foley catheter placement is mandatory to allow continuous evaluation of the urinary output as a guide for renal perfusion in shocked patients.
(8) Serum level of plasma cholinesterase, arterial blood gases, and ECG should be done to every suspected case with OP poisoning.
(9) All physicians should be aware of information about the use of atropine according to guidelines: give atropine 0.02 mg/kg intravenously, as per the regime a bolus loading dose s started, followed by boluses after a fixed time interval varying from 5 to 15 to 30 min till atropinization (clear chest on auscultation with no wheezing).
(10) The frequency of boluses of atropine should be limited by the appearance of signs of atropine toxicity such as confusion, agitation, hyperthermia, ileus, and tachycardia.
(11) Pralidoxime should be taken in all cases to reverse acetylcholinesterase inhibition.
(12) Benzodiazepines should be given intravenously in case of agitation or seizures at a dose starting with: diazepam (0.05–0.3 mg/kg/dose) and even in the absence of seizures to increase the survival and reduce CNS damage, and cardiac dysfunction.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

Financial support and sponsorship
Nil.

Conflicts of interest
There are no conflicts of interest.

References

1 Bruyndonckx RB, Meulemans AI, Sabbe MB, Kumar AA., Delooz HH. Fatal intentional poisoning cases admitted to the University Hospitals of Leuven, Belgium from 1993 to 1996. Eur J Emerg Med 2002; 9:238–243.

2 Leibson T., Lifshitz M. Organophosphate and carbamate poisoning: review of the current literature and summary of clinical and laboratory experience in southern Israel. Isr Med Assoc J 2008; 10:767–770.

3 Selvaraj T, Sudharson T. Demographic and clinical profile of organophosphorus poisoning cases in a Medical College Hospital, Tamil Nadu. Indian J Foren Comm Med 2016; 3:124–127.

4 Karalliedde L, Baker D, Marrs TC. Organophosphate-induced intermediate syndrome. Toxicol Rev 2006; 25:1–14.

5 Eddleston M, Singh S, Buckley N. Organophosphorus poisoning (acute). BMJ Clin Evid 2011; 1744:2102.

6 Eddleston M., Mohamed F., Davies JO., Eyer P., Worek F., Sheriff MR, *et al*. Respiratory failure in acute organophosphorus pesticide self-poisoning. J Assoc Physicians 2006; 99:513–522.

7 Eddleston M, Buckley NA, Eyer P, Dawson AH. Management of acute organophosphorus pesticide poisoning. Lancet 2008; 371:597–607.