ORIGINAL ARTICLE

A retrospective review of intensive care management of organophosphate insecticide poisoning: Single center experience

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Abstract

Background: Organophosphate (OP) compounds are used as insecticides. Given the widespread availability and use of these chemicals, OP poisoning is quite common following either accidental or intentional exposures. Immediate intensive care management can save lives in these patients. We aimed to investigate intensive care management provided to OP poisoning patients in a tertiary care hospital in Turkey.

Subjects and Methods: This was a retrospective chart review of 62 patients, admitted to the Intensive Care Unit (ICU) with OP poisoning between 2000 and 2012.

Results: Of the 62 patients studied, 40 (65%) were male, 45 (73%) were suicide attempts, 59 (95%) ingested the OP compounds, and three patients (5%) (two patients with suicide and 1 with accidental exposure) died in the ICU. There were statistically significant differences between survivors and nonsurvivors for Glasgow Coma Scale (GCS) on admission (P = 0.034), Acute Physiology and Chronic Health Evaluation II (APACHE II) score (P = 0.003), Sequential Organ Failure Assessment (SOFA) score (P = 0.024), time to initiation of treatment (P = 0.034) and serum lactate dehydrogenase (LDH) levels (P = 0.007).

Conclusions: Organophosphate poisoning is a life-threatening condition that requires immediate diagnosis and management. GCS, APACHE II score, SOFA score, and time to admission to the emergency department and LDH levels can provide prognostic information and predict outcomes.

Key words: Clinical outcomes, Intensive Care Unit, mortality, organophosphate poisoning

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Introduction

Insecticides have become essential to agriculture around the world over the last five decades.[1] Organophosphate (OP) compounds are commonly used as insecticides internationally due to their widespread availability, low cost, and relatively rapid degradation following application. Frequently used compounds have included malathion, parathion, chlorpyrifos, diazinon, dichlorvos, fenitrothion, tetrachlorvinphos, and azinphos-methyl.[2-4]

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Acetylcholine is a neurotransmitter present at the neuromuscular junctions in peripheral and central nervous systems. Acetylcholinesterase (AChE) is an enzyme that normally hydrolyzes and breaks down acetylcholine. OP compounds cause phosphorylation and inactivation of this enzyme leading to the accumulation of acetylcholine.[3]

One of the lethal complications following OP poisoning is the development of respiratory failure. This may occur due

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to many reasons, including aspiration of gastrointestinal contents, excessive secretions, neuromuscular involvement, intermediate syndrome, septicemia, and adult respiratory distress syndrome. Early recognition of respiratory failure, early endotracheal intubation, and mechanical ventilation are life-saving in severe OP poisoning.[5,6] These patients need intensive care management for respiratory and close hemodynamic monitoring due to above-mentioned reasons.

Previous studies have reported high mortality rates following OP poisoning, the majority of which could have been prevented by early diagnosis and treatment.[6-8] There is a significant improvement, in general, critical care of the patients in the last decade, and we would like to determine the effect of general critical care improvements on OP poisoning patients in our center. The aim of this study was to describe intensive care management provided to OP poisoning patients in a tertiary care hospital in Turkey.

Subjects and Methods

This retrospective study was performed on patients admitted with OP poisoning to the eighteen bed medical Intensive Care Unit (ICU) at the Erciyes University Hospital, between 2000 and 2012. The study was approved by Institutional Ethic Committee (Consent No.: 2013/15; Date 08.01.2013). Data from sixty-two patients were collected and analyzed. Data were collected from the patients' chart. Diagnosis of OP poisoning was based on information taken either from the patient or their family about the agent involved in the exposure. Gastric lavage, administration of activated charcoal via nasogastric tube, and cleansing of the patient with soap and water was started as soon as the patient arrived to the emergency department. The patients were admitted to the ICU based on the severity of the clinical signs and symptoms. We confirmed the diagnosis of OP poisoning by measuring plasma pseudocholinesterase (PCE) levels. PCE levels were determined using an Olympus AU2700 spectrophotometric chemistry analyzer (Beckman Coulter, Tokyo, Japan). However, even if PCE levels were found to be normal, but clinical symptoms strongly suggested acute OP toxicity and known exposure to OP agent, patients were treated with the OP poisoning treatment regimen. Treatment was started as soon as the diagnosis of OP poisoning was suspected. Atropine and/or pralidoxime sulfate was used. Atropine was given as a continuous infusion after a loading dose of 1 mg of atropine every 5 min up to 3 or 4 doses. Continuous infusion was started at 0.5–2 mg/h until control of the hypersecretion symptoms occurred. Heart rate and pupil size were not used as indices of atropine titration as long as the heart rate was above 60 beats/min. Atropine was discontinued 24 h after all signs of atropinization (facial flushing, dilatation of pupils, dryness of mouth, tachycardia) occurred. Continuous infusion of pralidoxime sulfate was administered at 4 mg/kg/h after a 1000 mg intravenous bolus injection for at least 24 h based on clinical status. Arterial blood gas and routine biochemistry were performed daily and as needed. The indications for endotracheal intubation and mechanical ventilation were as follows: Uncontrolled secretions; depression of consciousness, inability to protect the airway, hypoxia which was unresponsive to oxygen treatment, cardiopulmonary arrest, and metabolic acidosis with hemodynamic instability. Intubated patients received synchronized intermittent mandatory ventilation with pressure support in either pressure-controlled or volume-controlled form. Low tidal volume strategy was used for these patients. Positive end expiratory pressure was initially applied as 5 cm H_2O initially and then titrated according to oxygen saturation (SaO_2). Weaning from mechanical ventilation was carried out with pressure support mode and daily T-tube trials.

Demographic and routine laboratory results were recorded for the duration of the patient's stay in hospital. Data related to a number of clinical outcomes such as Glasgow Coma Scale (GCS) on admission to emergency department, Sequential Organ Failure Assessment (SOFA) score and Acute Physiology and Chronic Health Evaluation II (APACHE II) scores on entry to the ICU, duration of mechanical ventilation, and length of ICU and hospital stay were also recorded. PCE levels were measured daily in the ICU.

Statistical analysis

SPSS 16.0 software (SPSS Inc., Chicago, IL, USA) was used for the statistical analysis. Continuous variables with the normal distribution are presented as mean ± standard deviation median values were used where normal distribution was absent. Statistical analysis for the parametric variables was performed using the Student's *t*-test between the two groups. The Mann–Whitney U-test was used to compare nonparametric variables between the two groups. Qualitative variables are given as percentages and the correlation between categorical variables was investigated using the Chi-square test. $P < 0.05$ considered statistically significant.

Results

During the study period, 62 patients with OP poisoning were admitted to our ICU. Table 1 shows characteristics of 62 patients. The mean age for all patients was 39 ± 16 years. Of the 62 patients, 40 (65%) were male and 22 (35%) were female. The reason for poisoning was a suicide attempt in 45 (73%) patients and accidental exposure in 17 (27%) patients. Gastrointestinal system was the route of exposure in 59 (95%) of 62 patients. One (1.6%) patient had skin exposure and 2 (3.2%) were poisoned through more than one route with skin and gastrointestinal system. Table 2 shows OP insecticide agents responsible for the poisonings. There were twelve different types of OP agents. Type of OP compounds was not known in 10 patients. Three (5%) patients (two patient's suicide, one patient accidental exposure) died in

Table 1: Demografic and clinical characteristics of the patients

Parameters	
Age, mean (SD), years	39±16
Gender, *n* (%)	
Male	40 (65)
Female	22 (35)
Presence of co-morbidity, *n* (%)	18 (28.6)
History of psychiatric disorder, *n* (%)	10 (15.9)
History of suicide, *n* (%)	2 (3.2)
Marital status, *n* (%)	
Married	47 (76)
Single	15 (24)
Admitted from, *n* (%)	
Urban area	25 (40)
Rural area	37 (60)
Smoking, *n* (%)	27 (43)
Alcohol abuse, *n* (%)	9 (14.3)
Systolic blood pressure, mean (SD), mmHg	122±30
Diastolic blood pressure, mean (SD), mmHg	77±18
Heart rate, mean (SD), beats/min	91±27
Respiratory rate, mean (SD), breaths/min	22±3.9
Body temperature, mean (SD), °C	36±0.5

SD=Standard deviation

Table 2: Organophosphate insecticide agents responsible for the poisonings

Agent	*N*, (%)
Methamidophos	8 (13)
Ethvl-paration	7 (11)
Dichlorvos	7 (11)
Azinphos-methyl	5 (8)
Chlorpyrifos	4 (6)
Diazinon	4 (6)
Monocrotophos	4 (6)
Malathion	4 (6)
Methidathion	4 (6)
Coumaphos	2 (3)
Trichlorphon	2 (3)
Chlorphorvinphos	2 (3)
Unknown	10 (16)

Table 3: Clinical signs and symptoms

Signs and symptoms	*N* (%)
Muscarinic	
Nausea	30 (48)
Vomiting	26 (41)
Myosis	16 (25)
Drowsiness	15 (24)
Diarrhea	9 (14)
Hypersalivation	9 (14)
Bradycardia	7 (11)
Hypotension	6 (10)
Abdominal pain	5 (8)
Urinary incontinence	2 (3)
Nicotinic	
Fasciculation	2 (3)
Tachycardia	17 (27)
Hypertension	16 (25)
Central nervous system	
Coma	3 (5)
Altered consciousness	26 (41)
Convulsion	3 (5)
Headache	3 (5)
Agitation	9 (14)

Table 4: Laboratory values on admission

Parameters	
WBC, mean (SD), x10^9/L	14±6
Hemoglobin, mean (SD), g/dL	14±2
Platelet count, mean (SD),/mm^3	247857±73085
BUN, mean (SD), mg/dL	13±5
Creatinine, mean (SD), mg/dL	0.9±0.2
Sodium, mean (SD), mEq/L	139±4
Potassium, mean (SD), mEq/L	4±0.5
Calcium, mean (SD), mg/dL	8.9±0.7
Glucose, (range), mg/dL	120 (62-413)
AST, (range), (u/L)	27 (14-124)
ALT, (range), (u/L)	17 (9-84)
LDH, mean (SD), (u/L)	285±139

ALT=Alanine transaminase; AST=Aspartate transaminase; BUN=Blood urea nitrogen, LDH=Lactate dehydrogenase; WBC=White blood cell

the ICU. The median GCS on the admission to both the emergency department and ICU was 15.0 (range, 3–15). APACHE II score in the admission to ICU was 8.5 (range, 1–29). The median SOFA score was 1 (range, 0–12). The estimated median time for the admission to the emergency department and ICU after the OP exposure was 1.5 (range, 0.5–48) h and 13.0 (range, 5–120) h, respectively. The most frequent clinical findings were nausea, vomiting, unconsciousness, palpitation, and tachycardia [Table 3]. There were leukocytosis, hyperglycemia, and high levels of lactate dehydrogenase (LDH) on admission [Table 4].

The time from exposure to first intervention was 15 min in 8 (12.7%) patients, 1 h in 20 (32.3%) patients, 1–3 h in 22 (35.5%) patients, 3–6 h in 8 (12.9%) patients, 12–24 h 3 (4.8%) patients, and after the first 24 h in 1 (1.6%) patient.

All patients received atropine. Atropine was administered for a median of 5.0 days (range: 1–48) and median total atropine dose was 88.5 mg (range: 2–310). Pralidoxime was administered to 61 (98.4%) patients. Pralidoxime was given for a median of 3.2 days (range, 1–8) and median total pralidoxime dose was 15,515 mg (range: 500–33,600). Only 3 (5%) patients died. Table 5 shows clinical outcomes observed in the 62 patients. Median arterial blood gas values of these patients on admission were as follows: pH 7.40 (range: 7.10–7.54); PaO_2 98.6 mmHg (range: 47–220); $PaCO_2$ 33.5 mmHg

Table 5: Clinical outcomes observed in the 62 patients with organophosphate poisoning

Parameter	For all patients	Alive	Dead	*P*
GCS, (range)	15 (3-15)	15 (3-15)	6 (3-14)	0.034
APACHE II score, (range)	8.5 (1-29)	8 (1-25)	22 (21-29)	0.003
SOFA score, (range)	1.0 (0-12)	1.0 (0-8)	5.0 (3-12)	0.024
Time from exposure to emergency department admission, (range), hour	1.5 (0.5-48)	1.5 (0.5-48)	5.0 (2-5)	0.034
Time from emergency department admission to ICU transfer, (range), hour	14 (5-120)	13 (5-120)	30 (12-100)	0.157
ICU LOS, (range), day	5.5 (1-24)	5.5 (1-24)	4 (1.5-24)	0.596
Hospital LOS (range), day	7 (3.5-69)	7.0 (3.5-69)	4.5 (4-25)	0.511
Duration of mechanical ventilation, (range), days	4.0 (1-24)	4 (1-17)	13 (2-24)	0.641
White blood cell count, (range), ($x10^9$/L)	12.890 (5130-30280)	12900 (5130-30280)	11700 (11450-25700)	0.902
pH, (range)	7.40 (7.10-7.54)	7.40 (7.10-7.54)	7.30 (7.15-7.49)	0.294
$PaCO_2$, (range), mmHg	33.5 (21-58)	34.0 (21-58)	33 (31-54)	0.450
HCO_3, (range), mmol/L	22 (12-32)	22 (12-32)	25.6 (12-26.40)	0.641
PCE level, (range), U/L	450 (15.80-7377)	452 (39.70-7377)	88 (15.80-3815)	0.325
Lactate Dehydrogenase, (range), (u/L)	251 (117-955)	248 (117-955)	440 (381-733)	0.007
Intermediate syndrome, *N* (%)	4 (6.5)	3 (5.1)	1 (33.3)	0.052

APACHE=Acute Physiology and chronic health evaluation; GCS=Glasgow coma scale; ICU=Intensive care unit; LOS=Lenght of stay; PCE=Pseudocholinesterase; SOFA=Sequential organ failure assessment

Table 6: ECG findings in patients presenting with cardiac signs

Parameters	*n* (%)
Tachycardia	17 (27.4)
Bradycardia	8 (13)
ST elevation	4 (6.5)
Atrial fibrillation	3 (4.8)
Prolonged QT interval	2 (3.2)
T wave inversion	1 (1.6)

ECG=Electrocardiogram

(range: 21–58); HCO_3 22 mmol/L (range: 12.0–32.0); SaO_2 96.0% (range: 74.0–99.0%). Mechanical ventilator support and reintubations after extubation were needed for 18 (28.6%), 10 (15.9%) patients, respectively. The duration of mechanical ventilation was 4 days (range: 1–24). Intermediate syndrome developed in only 4 (6.5%) patients. The median length of stay in ICU and hospital was 5.5 (range: 1–24) and 7 days (range: 3.5–69), respectively.

Of the 62 patients studied, 27 (42.9%) patients developed infection during follow-up; pneumonia in 20 (32.3%), urinary tract infection in 3 (4.8%), soft tissue infection in 2 (3.2%), central venous catheter-related infection in 1 (1.6%), and oral candidiasis in 1 (1.6%) patient.

The most frequent electrocardiogram findings in patients presenting with cardiac signs were tachycardia, bradycardia, and sinus tachycardia elevation [Table 6].

The mean pseudocholinesterase level of the patients on admission was 340 U/L. This level gradually increased during the follow-up period. Patient's mean pseudocholinesterase levels are shown in Figure 1.

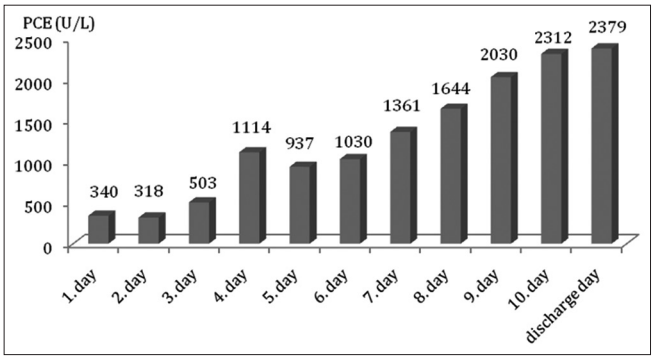

Figure 1: Pseudocholinesterase (PCE) levels of the patients (U/L)

Discussion

Organophosphate poisoning is quite common in the developing world due to the extensive use and uncontrolled accessibility of these compounds. Accidental and intentional OP poisoning is commonly seen in Turkey, especially in rural areas, and OP ingestion has been found to be a commonly used means to commit suicide.[6,7,9] The reason for poisoning was a suicide attempt in 73% of the patients. This finding is not unexpected given the fact that 60% of our study population resided in rural areas where the majority worked in the agricultural industry and therefore had easy access to pesticides.

Organophosphate poisoning commonly occurs following ingestion, inhalation, and absorption of OP compounds.[7,10] Given that the majority of OP pesticides are liquid formulations, the most common and easiest mode of exposure is via oral ingestion. The vast majority of patients, 59 (95%) ingested the OP pesticides in our study.

Of the cases studied, 65% were male. A number of studies have made similar observations related to the higher incidence of OP poisoning in males.[10,11] This may be explained by the fact that individuals working in the agricultural sector are predominantly male; therefore they are more likely to be exposed to OP pesticides.

The WHO pesticide hazard class (I, II, or III), is a graded system dictates that the nature and extent of clinical manifestations and toxicity profile with class I being the most toxic and III the least.[12] In our study population, the most common OP compound causing poisoning was methamidophos (8%), dimethyl, class I pesticide. Ethvl-parathion (a diethyl, class I pesticide) and dichlorvos (a dimethyl, class I pesticide) were each the cause of poisoning in 7% of the patients studied.

Of the three patients who died, one was poisoned with parathion, and another was caused by chlorpyrifos (diethyl, class II pesticide). The third patient died from an unknown OP compound.

The symptoms observed following OP poisoning are meiosis, seizures, glandular hypersecretion, vomiting, diarrhea, bradycardia, and neuromuscular dysfunction.[3] Patients with severe acute OP poisoning can develop respiratory failure which can be life-threatening.[13] The most common symptoms observed in our patient population while in the ICU were muscarinic in nature such as nausea (48%) and vomiting (41%). Nicotinic symptoms such as tachycardia and hypertension were also observed in about a quarter of our study population.

Of the cases studied, 41% presented to the emergency department with altered consciousness, and the GCS was used to access neurological function. GCS is a well-recognized scoring system which has been found to be a reliable and objective way to evaluate brain function and predict neurological outcomes.[14] Bilgin *et al*. conducted a study in ICU patients to compare the ability of three scoring systems (GCS, APACHE II, and SAPS II) to predict mortality in patients following OP poisoning. They found that all three had similar predictive abilities; however, they concluded that GCS system has superiority over the others as it was easy to perform, and did not require complex physiologic parameters and laboratory methods.[15] Cander *et al*. showed that GCS values were effective in predicting the outcomes wherein patients with a median GCS of 3 died while all of those with a score of 15 were discharged.[16] In our study, median GCS of patients who died was 6 while the score for those who survived was 15, which is similar to what other studies have shown.

Another prognostic assessment tool that has been used extensively around the world is the APACHE II system.[17] This ICU scoring systems can be used to measure and describe the severity of disease, the morbidity and prognosis of patients. Lee and Tai evaluated the ability of the APACHE II score to predict mortality in patients with OP poisoning and found that a score of 26 or higher was associated with a higher risk of death.[18] Kang *et al*. found that the APACHE II score was significant predictor of mortality (odds ratio [OR], 1.194 95% confidence interval [CI]: 1.089–1.309) and respiratory failure (OR, 1.273 95% CI: 1.122–1.444).[19] We found that patients who died had a median APACHE II score of 22 while those who survived that a median score of 8, thus corroborating the results of previous studies.

The SOFA scoring system is a widely used means to track a patient's organ failure status during ICU stay by determining the extent of a patient's organ function or rate of failure.[20] The score is based on six physiological systems including the respiratory, cardiovascular, hepatic, coagulation, renal, and neurological systems. A study of OP poisoning patients in the ICU performed by Lee *et al*. found that the mean SOFA score of survivors was 3.9 ± 2.9 and 6.7 ± 2.7 for nonsurvivors.[21] The patients who died in our study had a median SOFA score of 5 while the score of those who survived had a median score of 1.

A critical factor found to influence the mortality rate is the time to treatment initiation following exposure to OP pesticides.[9] The median time from exposure to the emergency department admission was 5.0 h among patients who died, while the median time was 1.5 h for patients that survived in our study. Early treatment may be very important to reduce mortality.

Another potential prognostic indicator for OP poisoning cases could be LDH levels. We found out that the LDH levels were significantly different in survivors (248 u/L [range, 117–955]) compared to the nonsurvivors (440 u/L [range, 381–733]) $P = 0.007$. High LDH levels may be caused by OP-induced oxidative tissue damage[22] or muscle injury.[23]

Intermediate syndrome is a state of muscle paralysis that occurs in the interval between the end of the cholinergic crisis and before the onset of OP induced delayed polyneuropathy. Symptoms of this syndrome manifest within 24–96 h after exposure.[24] Etiology, incidence, and risk factors are not clearly understood, but it is accepted as a disorder of neuromuscular junctions.[25] Patients with intermediate syndrome require respiratory support, atropine, and pralidoxime. Of the patients studied, 4 suffered from intermediate syndrome, all of whom required mechanical ventilation and 2 required re-intubation after being weaned and extubated. One of the four patients died due to respiratory failure, Clogging of the tubing of the mechanical ventilator is quite common, necessitating a higher than

expected rate of reintubations due to the massive amounts of secretions produced following OP poisoning. This may explain the high rate of reintubations observed in our study population.

The most commonly used technique to diagnose OP poisoning is determination of PCE (butyrylcholinesterase) levels in the plasma or red blood cell.[3,26] Chen *et al*. found that the absence of rising PCE levels in the 48 h after treatment of OP poisoning was associated with a higher mortality rate.[27] Another study conducted by Tsai *et al*. found no significant association between the severity of OP poisoning and serum AChE levels.[28] A study conducted by Manu *et al*. found that serial measurements of serum AChE levels may help predict the length of ICU stay, duration on mechanical ventilation and the prognosis of the patient following OP poisoning.[29] There was no statistically significant difference in the PCE levels among patients that survived versus those that died in our study population.

Poisoning due to OP is an important cause of morbidity and mortality.[30] Mortality rate associated with OP poisoning was found to be between 28% and 47% in the period between 1980 and 2000.[6-8] This may be explained by the fact that atropine was given at a very low dose for a short time with pralidoxime not prescribed to all patients and insufficient supportive therapy. Studies conducted after the year of 2000 have consistently shown mortality rates below 15%.[11,31,32] This can be explained by universal provision of continuous high dose atropine, consistent use of pralidoxime and better access to supportive therapy such as ICU care, ventilator support, respiratory therapy, and hemoperfusion.[28,32-35]

Conclusions

Organophosphate poisoning is a life-threating condition that requires immediate diagnosis and treatment. Early initiation of atropine and pralidoxime therapy, with supportive ICU care, can save lives. GCS on admission to the emergency department, APACHE II score for the first 24 h in the ICU, SOFA score, and time from exposure to initiation of treatment and serum LDH levels can provide useful prognostic information and help predict outcomes.

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