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원 저

유기인계 중독에 의한 심근손상 환자에서의 경흉부 심장 초음파검사를 사용한 심장기능평가

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Evaluation of Cardiac Function by Transthoracic Echocardiography in Patients with Myocardial Injury Secondary to Organophosphate Poisoning

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Purpose: Cardiac complications may occur in cases of organophosphate (OP) poisoning. However, a few studies regarding patterns of cardiac toxicity as determined by transthoracic echocardiography (TTE) after exposure to OP have been reported. In the current study, the authors examined cardiac functions using TTE in patients with myocardial injury caused by exposure to OP.

Methods: A retrospective review was conducted on 16 consecutive cases of OP poisoning with myocardial injury (defined as elevated troponin I within 48 hours of arrival at the regional emergency center in South Korea and diagnosed and treated at the center from January 2012 to November 2014.

Results: TTE was performed in 11 (69%) of the 16 patients with an elevated troponin I (TnI) level within 48 hours. Of these 11 patients, 5 patients (45.5%) exhibited reduced ejection fraction (EF), and 3 exhibited regional wall motion abnormality (RWMA). Two patients (18.2%) had both reduced systolic function and RWMA. Two of the 5 patients with reduced EF returned to normal systolic function, however two patients did not regain normal systolic function after admission. One patient expired due to multiple organ failure, and 4 patients were transferred with a moribund status. Twelve of 15 patients who survived to discharge (at 4 to 35 months) were followed. Five of these patients died during follow-up and 7 survived without further complications.

Conclusion: OP can cause reversible cardiac dysfunction including reduced systolic function and RWMA. Serum TnI may be useful for initial assessment of cardiac function during the workup of patients suffering from OP poisoning. After the initial assessment of cardiac enzyme, further evaluation with TTE in patients with abnormal cardiac enzyme will be necessary to understand the cardiac toxicity.

Key Words: Organophosphate, Echocardiography, Troponin I, Electrocardiogram, Poisoning

Introduction

Organophosphates (OPs) are the most widely used insecticides worldwide, and poisoning by these compounds is an especially important environmental problem in developing countries[1]. Organophosphate (OP) insecticides irreversibly inhibit acetyl-

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cholinesterase, which causes acetylcholine accumulation, and over-stimulating cholinergic synapses in the central nervous system, somatic nerves, parasympathetic nerve endings, and sweat glands[2,3]. Furthermore, cardiac complications, such as, various arrhythmias, conduction disturbances, hypertension-hypotension, and myocardial injury, have been reported after OP poisoning[4-7]. Generally, these cardiac complications are classified, as originally described by Ludomirsky et al.into three phases, that is, an initial intense sympathetic phase, a prolonged parasympathetic phase, and a QT prolongation phase. In addition, there is a possibility of direct myocardial injury due to the cardio-toxic effect of OP[7-9].

However, few reports have been issued regarding patterns of cardiac toxicity, especially by transthoracic echocardiography (TTE), after exposure to OP[9,10]. To be familiar with changes in cardiac function in patients with myocardial injury, we investigated the cardiac function using, EKG, troponin I, and TTE in patients with OP-induced myocardial injury.

Methods

1. Study design and data

This retrospective observational study was conducted on 16 consecutive patients with acute OP poisoning that presented with myocardial injury, defined as an elevated troponin I (TnI) level, within 48 hours of arrival at the regional emergency department (ED) in South Korea, between January 2012 and November 2014. The exclusion criteria applied were; age 〈18 years, poisoning by any additional agent (with the exception of alcohol), acute coronary syndrome, end stage renal disease, no elevation of TnI within 48 hours, and cardiac arrest at ED arrival. Poisoning with OP was reported by patients or guardians. The identity of the toxic agent was confirmed by an emergency physician, who transcribed the bottle label into patient records. Levels of serum pseudocholinesterase were also measured for diagnostic purposes.

Data were retrospectively collected from medical records and reviewed. The following parameters were assessed: age, gender, amount of OP ingested, class of OP based on the WHO classification scheme, cause of poisoning, time elapsed from ingestion to ED arrival, route of poisoning, initial Glasgow Coma Scale (GCS), electrocardiogram (ECG), initial vital signs, and TTE, which was performed immediately

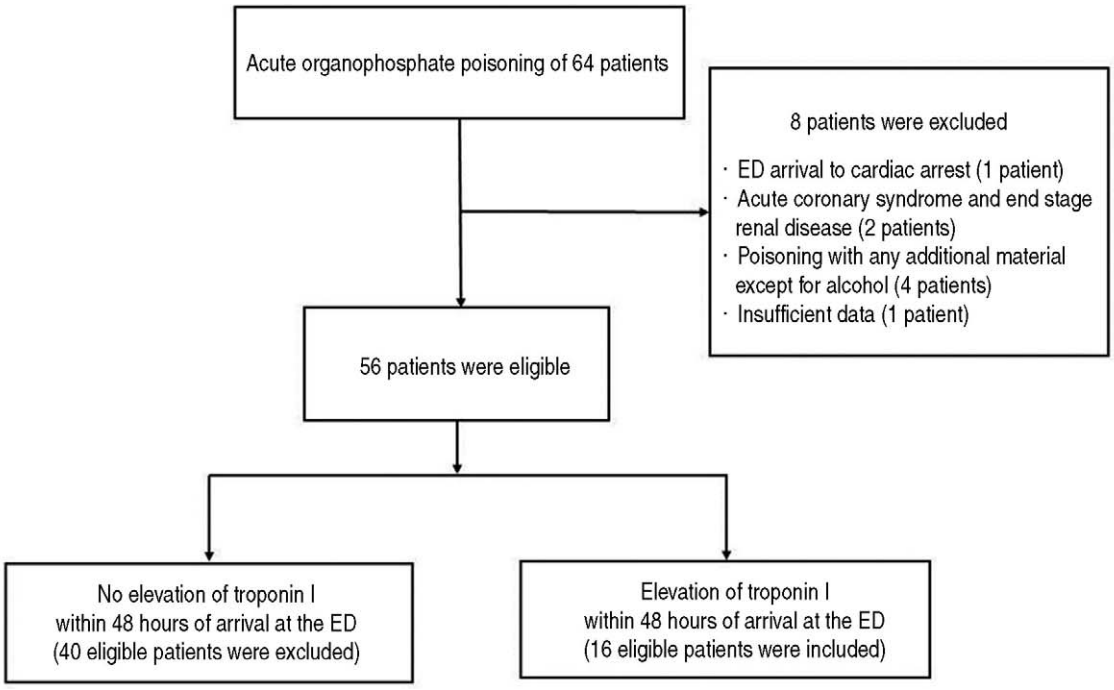

Fig. 1. Description of numbers of acute organophosphate poisoning patients assessed, excluded, and included in a study.

after TnI elevation was recognized[11]. We assessed atropine dosage for 48 hours as this drug may affect the heart due to atropine induced tachycardia. Arterial blood gas testing was performed and serum lactate levels were measured. Cardiac biochemical markers including creatine kinase MB (CK-MB), TnI (Siemens Healthcare Diagnostics Inc., Newark, DE, USA), and β-natriuretic peptide (BNP) were investigated in the

Table 1. Characteristics and laboratory findings of patients with myocardial injury resulting from acute organophosphate poisoning

Characteristics	Myocardial injury (n=16)
Age (years)	61±17*
Male	10 (62.5%)
Volume (ml)	150 (100-300)†
Oral route	16 (100%)
Intentional poisoning	14 (93.3%)
Time to ED arrival (minutes)	117 (60-327)†
Co-ingested with alcohol	4 (28.6%)
Class of organophosphate	
Class I	4 (25.0%)
Class II	3 (18.8%)
Class III	0 (0%)
Unclassified	9 (56.3%)
Initial SBP (mmHg)	130±54*
Initial GCS	7.0 (3.0-8.8)†
Initial hypoxia (PaO_2<60 mmHg)	3 (18.8%)
Initial shock (SBP<90 mmHg)	5 (31.3%)
Gastric lavage	7 (43.8%)
Activated charcoal	16 (100%)
Use of pralidoxime	16 (100%)
Use of atropine	16 (100%)
Atropine dosage for 48 h (mg)	60.0 (27.6-116.3)†
Laboratory findings	
Pseudocholinesterase (U/L)	300 (300-487)†
pH	7.30 (7.14-7.35)†
PO_2 (mmHg)	99.8 (78.0-138.1)†
PCO_2 (mmHg)	31.9 (26.9-45.9)†
HCO3 (mmol/L)	16.4 (12.9-19.5)†
Lactate (mmol/L)	5.70 (2.90-8.62)†
Namba classification	
Mild	0 (0%)
Moderate	0 (0%)
Severe	16 (100%)
Complications	
Respiratory failure requiring mechanical ventilation	16 (100%)
Shock (SBP<90 mmHg)	7 (43.8%)
Use of adrenergic agents	7 (100%)
Pneumonia	9 (56.3%)
Decreased mental status (GCS<13)	15 (93.8%)
Outcomes	
Total admission days	22 (8-33)†
ICU admission days	17 (3-21)†
Mortality	1 (6.3%)

* Mean±Standard deviation

† Median (interquartile range)

ED: emergency department, SBP: systolic blood pressure, GCS: Glasgow Coma Scale, ICU: intensive care unit

Table 2. General characteristics of individual patients with myocardial injury resulting from acute organophosphate poisoning

Patient Number	Age /Sex	Route	Volume (cc)	Classification of OP	Intention	EDAT	Co-ingestion with alcohol	Previous Alcohol history	Gastric lavage	Charcoal	Atropine Dosage for 48 h (mg)	SBP	Initial GCS	Namba classification
1	49/M	Oral	50	II (chloropyrifos)	Yes	210	No	Daily 1 bottle	No	Yes	50.5	121	9	Severe
2	29/F	Oral	300	Unclassified	Yes	60	No	None	Yes	Yes	40.5	101	15	Severe
3	47/F	Oral		Unclassified		70		None	No	Yes		139	11	Severe
4	39/F	Oral	150	I (phosphamidon)	No	70	No	None	Yes	Yes	11.5	122	13	Severe
5	63/M	Oral	15	Unclassified	Yes	200		Daily 1 bottle	No	Yes	26.4	87	6	Severe
6	69/F	Oral		Unclassified	Yes	480	No	Unknown	No	Yes	27.6	82	3	Severe
7	46/F	Oral	200	I (monocrotophos)	Yes	155	No	None	Yes	Yes	116.3	58	8	Severe
8	64/M	Oral		Unclassified	Yes	465	Yes	Quit for 10 yrs	No	Yes	21.0	148	7	Severe
9	80/F	Oral	100	II (fenitrothion)	Yes	50	No	Unknown	No	Yes	53.3	229	7	Severe
10	58/M	Oral	300	II (dichlorvos)	Yes	355	Yes	Quit for 2 yrs	Yes	Yes	220.0	73	7	Severe
11	68/M	Oral	300	Unclassified	Yes	75	No	2~3/month Half bottle	No	Yes	130.5	221	3	Severe
12	91/M	Oral	100	Unclassified	Yes	45	No	None	Yes	Yes	100.5	153	3	Severe
13	51/M	Oral		Unclassified	Yes	420	Yes	Unknown	No	Yes	100.5	94	3	Severe
14	71/M	Oral	100	I (phosphamidon)	Yes	245	No	Daily 2~3 bottles	Yes	Yes	60.5	194	6	Severe
15	76/M	Oral		Unclassified	Yes	60	No	None	No	Yes	60.0	78	8	Severe
16	76/M	Oral	500	I (monocrotophos)	Yes	60	Yes	4/week 1~2 bottles	Yes	Yes	120.0	183	3	Severe

OP: organophosphate, EDAT: emergency department arrival time after ingestion, SBP: systolic blood pressure, GCS: Glasgow Coma Scale

ED. Corrected QT intervals (QTc) determined by ECG were calculated using Bazett' s formula, and a prolonged QTc interval was defined one of more than 440 ms[12]. We investigated whether patients with an elevated TnI exhibited abnormal cardiac function using TTE to evaluate systolic function and regional wall motion abnormality (RWMA). Complications were defined as respiratory failure requiring mechanical ventilation, shock (systolic blood pressure 〈90 mmHg), pneumonia, decreased mental status (GCS〈13), and death. The study was approved by the institutional review board committee.

2. Statistical analysis

Statistical analyses were performed using SPSS for Windows (V.20.0 K, SPSS, Chicago, IL, USA). Nominal data are presented as frequencies and percentages, and continuous variables as means and standard deviations (SD) or as medians and inter-quartile ranges (IQR), after determining normality using the Shapiro-Wilk test. Statistical significance was accepted for p values 〈0.05.

Results

Sixty-four consecutive OP poisoned patients were treated during study period. However, 8 patients were excluded for the following reasons: ED arrival in a state of cardiac arrest (1 patient), acute coronary syndrome and end stage renal disease (2 patients), poisoning by an additional material except for alcohol (4 patients), and insufficient data (1 patient). In addition, 40 of the remaining 56 patients were excluded for no TnI elevation within 48 hours of arrival at the ED. Finally, 16 patients were included and these patients constituted the study cohort (Fig. 1). Between 2012 and 2014, total numbers of patients seen in our hospital' s ED ranged from 38,715 to 41,452 patients

annually.

Ten of the 16 patients were men (62.5%), and the ages of patients ranged from 29 to 91 years with a mean of 61±17 years. All patients were exposed via the oral route and the median ingested volume was 150 mL. Fourteen patients (93.3%) had been intentionally exposed to OP. All patients were treated with pralidoxime and atropine. The absorbed OPs included chloropyrifos, dichlorvos, fenitrothion, phosphamidone, monocrotophos, and others. Four (25.0%) and 3 patients (18.8%) were poisoned by Class I or II OP, respectively. According to the Namba classification of OP poisoning, all 16 patients exhibited severe toxicity (Table 1, 2)[13].

Median serum pseudo-cholinesterase at presentation was 300 U/L. Three patients (18.8%) presented with initial hypoxia (PaO_2〈60 mmHg) and 5 (31.3%) with initial shock (systolic blood pressure〈90 mmHg). Complications other than cardiac toxicity included; respiratory failure (16 patients, 100%), shock (systolic blood pressure 〈90 mm Hg) (7 patients, 43.8%), pneumonia (9 patients, 56.3%), and decreased mental status (15 patients, 93.8%). The median total admission days and ICU admission days were 22 and 17 days, respectively (Table 1, 3, 4).

Analysis of ECG performed ED revealed ST depression and ST elevation in 5 patients (33.3%) and 1 patient (6.7%), respectively. QT prolongation was present in 10 patients (62.5%). Cardiac biochemical marker analysis showed a median initial TnI level of 0.062 ng/ml. TTE was performed in 11 (69%) of the 16 patients with an elevated TnI within 48 h, and of these 11, 5 patients (45.5%) exhibited reduced ejection fraction (EF) and 3 (27.3%) exhibited RWMA. Two patients (18.2%) had both reduced systolic function and RWMA. Two of the 5 patients with reduced EF returned to normal systolic function and three of the 5 patients with reduced EF received inotropic treatment to support cardiac function. Two patients did not regain normal systolic function after admission (Table 5, 6).

One patient expired due to multiple organ failure, and 6 patients were transferred to another hospital. Twelve of the 15 patients that survived to discharge were followed for 4 to 35 months. Five patients died during follow-up and 7 survived without any further complications (Table 1, 4).

Discussion

Only few studies have evaluated cardiac dysfunction after exposure to OP[9,10]. In the present study, we

Table 3. Laboratory findings of individual patients with myocardial injury resulting from acute organophosphate poisoning

Patient Number	PSE	pH	pO2 (mmHg)	pCO2 (mmHg)	HCO3 (mmol/L)	Saturation (%)	Lactate (mmol/L)	Elevation time of TnI	Initial TnI	CK-MB	BNP
1	300	7.360	99.7	29.2	16.1	98.1	2.60	F/U	0.018	4.21	
2	300	7.423	61.8	32.4	20.7	92.8	1.61	F/U	0.015		
3	300	7.351	96.2	31.3	16.9	97.5	6.05	F/U	0.015	0.67	
4	418	7.325	87.5	38.7	19.7	95.8	5.73	F/U	0.015	0.50	
5	307	7.326	51.3	26.9	16.6	93.1	5.70	Initial	0.082	11.87	99.41
6	300	7.067	99.8	45.7	12.9	95.2	8.30	Initial	2.490	33.00	35.18
7	300	7.324	180.6	24.9	12.7	99.4	11.76	Initial	0.063	2.22	
8	739	7.165	128.6	18.4	6.5	98.1	11.72	Initial	0.340		
9	611	7.448	113.2	33.8	22.9	98.5	1.76	Initial	0.170	3.15	873.46
10	300	7.084	165.2	27.0	7.9	98.8	5.05	Initial	0.060		
11	510	7.272	87.2	30.4	13.7	94.4	5.70	Initial	0.065	1.06	36.74
12	300	6.782	54.6	88.4	12.9	56.3	11.27	F/U	0.015		
13	300	7.186	141.3	58.7	21.7	98.0	2.97	Initial	0.747	6.89	7.97
14	300	7.229	74.9	45.9	18.7	91.6	2.88	Initial	0.066	4.47	28.04
15	7368	7.331	122.1	25.6	13.2	97.6	8.72	F/U	0.015	1.17	28.48
16	300	7.131	185.4	51.4	16.8	98.7	5.61	F/U	0.024	11.62	62.54

PSE: pseudocholinesterase, TnI: troponin I, CK-MB: creatine kinase MB, BNP: B-type natriuretic peptide

Table 4. Clinical course of individual patients with myocardial injury resulting from acute organophosphate poisoning

Patient number	Respiratory failure	Pneumonia	Decreased mental status	Shock	Total admission days	ICU admission days	Outcome	Cause of death	Follow up
1	Yes	Yes	Yes	No	5	3			Alive with no problem
2	Yes	No	No	No	16	4			Alive with no problem
3	Yes	No	Yes	No	30	30	Transfer out (moribund discharge)	MOF	Death
4	Yes	No	Yes	No	20	12			Not contact
5	Yes	Yes	Yes	Yes	33	16			Alive with no problem
6	Yes	Yes	Yes	Yes	45	15			Alive with no problem
7	Yes	No	Yes	Yes	33	18			Alive with no problem
8	Yes	No	Yes	No	1	1	Transfer out (moribund discharge)	MOF	Death
9	Yes	No	Yes	No	19	19	Transfer out	Unknown	Death
10	Yes	Yes	Yes	Yes	2	2	Death	MOF	Death
11	Yes	No	Yes	No	18	18	Transfer out (moribund discharge)	MOF	Death
12	Yes	Yes	Yes	No	23	23	Transfer out (moribund discharge)	MOF	Death
13	Yes	Yes	Yes	Yes	1	1	Transfer out		Alive with no problem
14	Yes	Yes	Yes	Yes	33	21			Not contact
15	Yes	Yes	Yes	Yes	48	20			Alive with no problem
16	Yes	Yes	Yes	No	33	33			Not contact

ICU: intensive care unit, MOF: multiple organ failure

investigated the TTE findings of patients with TnI elevation within 48 hours of arrival at the ED to better characterize the effects of OP on the heart. Five patients (patients 5, 7, 9, 11, and 13) showed decreased ejection fraction on TTE, and 2 of these (patients 7 and 9) also showed RWMA. Follow-up TTE was performed in 4 patients (patients 5, 7, 9, and 11) to determine whether ejection fraction had recovered, and this was found to be so in patients 5 and 11 but not in patients 7 and 9, which both maintained a reduced ejection fraction and RWMA (Table 6). However, patient 7 survived for 24 months without any signs or symptoms of heart failure. We believe that in this female patient, reduced ejection fraction recovered to within the normal range, since she would otherwise have likely suffered from dyspnea or other symptoms resulting from reduced systolic function. Patient 9 (also a female patient) died in other hospital after discharge, although we do not know the cause of death (Table 4). Based on our results, we believe OP may have caused the reversible cardiac dysfunction.

In ANAND et al, they performed TTE on all OP poisoning patients regardless of elevation of TnI level and found systolic function to be normal in all[9]. However, in the present study, there were patients with reduced systolic function or RWMA. Even though all patients included had severe poisoning and an elevated TnI level in this study, the two studies differ in terms of reduced EF, which was not observed in any patient by ANAND et al[9].

He et al, reported TTE revealed marked decreases in wall motion of the inter-ventricular septum in all patients and of the left ventricle during the acute phase (this returned to normal during the recovery phase), and a significant improvement in left ventricular mean ejection fraction from 42% to 59% (p=0.001)[10]. However, in the present study, 45.5% of patients had reduced EF, 3 patients (27.3%) had RWMA, and 1 patient had only RWMA in the septum (patient 6) (Table 6). However, we included many OP types,

whereas only dichlorvos poisoning was included by He et al., which might explain this difference. Patient 10 had dichlorvos poisoning, but TTE was not performed on this patient. A well designed prospective TTE study is needed in many types of OP poisoning.

Although hypoxia and shock can affect myocardial injury, patients 7, 9, 11, and 13 had reduced EF without initial hypoxia and patients 9, 11, and 13 had reduced EF without initial shock, which indicates systolic function is not only reduced by hypoxia and shock (Table 2, 3, 6). In our opinion, systolic function is probably reduced directly by OP poisoning during the acute phase[14].

Atropine was administered as per our hospital's protocol, with an initial intravenous bolus of 1 to 3 mg depending on a severity of symptom. Continuous infusions were needed in patients suffering from severe poisoning by a highly fat-soluble OP, which continues to redistribute within fatty tissues. In the present study, all patients with reduced EF (patients 5, 7, 9, 11, and 13) had an elevated TnI before atropine was administered, and atropine infusion was discontinued when pulse rate exceeded 120 bpm to avoid myocardial injury secondary to tachycardia. Thus, we minimized the possibility of subsequent myocardial injury due to atropine use.

Four of the five patients with reduced EF (80%) exhibited simultaneous ECG changes consistent with ischemia, which included ST depression (Table 6). In a previous study, many OP poisoned patients had an elevated TnI level without ischemic changes by ECG[7]. Based on these results, we suggest cardiac function be evaluated by TTE when an OP poisoned patient exhibits ischemic change by ECG.

Table 5. Characteristics of ECG, cardiac markers, and TTE in patients with myocardial injury resulting from acute organophosphate poisoning

Characteristics	Myocardial injury (n=16)
ECG	
Rate	
Normal sinus rhythm	7 (43.8%)
Sinus tachycardia	8 (50.0%)
Sinus bradycardia	1 (6.3%)
ST and T wave change	
Normal	9 (60.0%)
ST depression	5 (33.3%)
ST elevation	1 (6.7%)
QT prolongation (>440 ms)	
Normal	6 (37.5%)
Prolongation	10 (62.5%)
PR prolongation (>120 ms)	
Normal	12 (92.3%)
Prolongation	1 (7.7%)
Cardiac markers	
Initial troponin I (ng/ml)	0.062 (0.015-0.148)*
Initial CK-MB (ng/ml)	3.68 (1.09-10.44)*
Initial BNP (pg/ml)	35.96 (28.15-90.19)*
Transthoracic echocardiographic findings (n=11)	
Systolic function	
Normal	6 (54.5%)
Reduced	5 (45.5%)
Wall motion abnormality	
Yes	3 (27.3%)
Reversibility	
Yes	2 (40%)

* Median (interquartile range)

ECG: electrocardiogram, CK-MB: creatine kinase MB, BNP: B-type natriuretic peptide

Table 6. TTE and ECG findings of patients with myocardial injury resulting from acute organophosphate poisoning

Patientnumber	ECG finding	QTc interval (ms)	TTE	EF	RWMA	Cardiac supportive treatment	Follow up TTE
1	Sinus tachycardia	475	Not performed			No	
2	NSR	413	Yes	Normal		No	
3	Sinus tachycardia	366	Not performed			No	
4	Sinus tachycardia	463	Yes	Normal		No	
5	NSR	418	Yes	Reduced		Inotropics	Recovered
6	Sinus tachycardia	576	Yes	Normal	Dyskinesis of apicoseptum	Inotropics	
7	NSR, ST depression	475	Yes	Reduced	Akinesis of posterior wall from base to mid	Inotropics	Not recovered
8	NSR	595	Yes	Normal		No	
9	NSR, ST depression	482	Yes	Reduced	Hypokinesis of posterolateral wall from base to mid	No	Not recovered
10	NSR, ST elevation	532	Not performed			Inotropics	
11	Sinus tachycardia, ST depression	519	Yes	Reduced		No	Recovered
12	Sinus tachycardia, ST depression	589	Not performed			No	
13	Sinus bradycardia, ST depression	541	Yes	Reduced		Inotropics	Not performed
14	Sinus tachycardia	397	Not performed			Inotropics	
15	Sinus tachycardia	524	Yes	Normal		Inotropics	
16	NSR	469	Yes	Normal		No	

ECG: electrocardiogram, NSR: normal sinus rhythm, TTE: transthoracic echocardiography, EF: ejection fraction, RWMA: regional wall motion abnormality, WMSI: wall motion score index

Clinical and laboratory signs have related the chronic alcoholism and cardiomyopathy[15]. Among the patients with myocardial injury, only four patients were recorded as co-ingested-with alcohol. However, only the amount and the frequency of alcohol consumption were recorded as the previous alcohol history. To understand the patient's status of alcohol usage or dependency, the psychological evaluation or analyzing the carbohydrate-deficient transferrin as a marker for diagnosis of chronic alcoholism, to detect the alcohol usage might be necessary[16,17]. For future studies, following up patients to evaluate the alcohol usage will be helpful to evaluate the cause of cardiomyopathy among patients with poisoning.

We attempted to contact patients or guardians to determine patient's current status; including survival, neurologic deficits after discharge, newly developed symptoms: such as dyspnea, chest pain, or chest discomfort, and any newly diagnosed cardiopulmonary conditions, such as heart failure or acute coronary syndrome after discharge. Most patients that survived to discharge were successfully contacted. Except patients 3, 8, 9, 11, and 12, the rest of patients were contacted and confirmed to have survived and to have suffered no further complications. Among the patients with cardiac dysfunction (patients 5, 7, 9, 11, and 13), two (patients 9 and 11) had expired (40%) (Table 4).

This study has several limitations. First, due to the small number of patients studied, strong conclusions cannot be drawn from our data; therefore, a larger scale prospective

study is necessary. Second, it is limited by its retrospective and observational design, and as a result, not all relevant assessment parameters could be included. In particular, volumes of OPs ingested and times elapsed between ingestion and ED arrival may have been over or underestimated. Third, since the percentages of OP, solvent, and surfactant in the various OP formulations were unknown, their clinical influences could not be determined.Forth, evaluation of status of alcohol usage will be necessary. In our opinion, well designed prospective TTE study is required to evaluate the effect of cardiac dysfunction on survival for poisonings with different OP types.

OP can cause reversible cardiac dysfunctions, including reduced systolic function and regional wall motionabnormalities. Even though the TTE study is useful for understanding the cardiac dysfunction, initial serum TnI may be necessaryto evaluate a cardiac function during the workup of patients suffering from OP poisoning.

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