Case Report

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Low-Dose Atropine-Triggered Atrial Fibrillation in Chlorpyrifos Plus Cypermethrin Poisoning: A Rare Case Report and Review of Literature

Farzad Gheshlaghi[1], Gholamali Dorooshi[1], Shiva Samsam-Shariat[2], Nastaran Eizadi-Mood[1], Leila Etemad[3], Pedram Pirmoradian[1] and Mohammad Moshiri[4,5]*

1. Clinical Toxicology Research Center, Isfahan University of Medical Sciences, Isfahan, Iran
2. Department of Clinical Toxicology, Isfahan Clinical Toxicology Research Center, School of Medicine, Isfahan University of Medical Sciences, Isfahan, Iran
3. Pharmaceutical Research Center, Pharmaceutical Technology Institute, Mashhad University of Medical Sciences, Mashhad, Iran
4. Medical Toxicology Research Center, Faculty of Medicine. Mashhad University of Medical Sciences, Mashhad, Iran
5. Department of Clinical Toxicology and Poisoning, Imam Reza Hospital, Faculty of Medicine. Mashhad University of Medical Sciences, Mashhad, Iran

*** Corresponding author**

Mohammad Moshiri, MD, PhD
Medical Toxicology Research Center, Faculty of Medicine, Mashhad University of Medical Sciences, Mashhad, Iran,
Department of Clinical Toxicology and poisoning, Imam Reza Hospital, Faculty of Medicine, Mashhad University of Medical Sciences, Mashhad, Iran
Tel: +98 9153416590, 51 3859 8973
Email: Moshirim@mums.ac.ir, Moshirimo@gmail.com

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Abstract

Background: Poisoning with Organophosphates (OP) and/or Pyrethroids (PYR) pesticides is common. We present a rare case of OP+PYR poisoned patient who suffered from Atrial Fibrillation (AF) at the beginning of treatment by a low dose of atropine and reviewed the literature.

Case Presentation: A 50-year-old man had ingested about 5-10 *ml* of a mixture of chlorpyrifos/cypermethrin. Half an hour later, he went to the rural hospital and 2 *hr* later, after gastrointestinal decontamination, he was referred to the clinical toxicology department with normal vital signs except normal sinus tachycardia [Heart rate (HR)]=105. On admission, he had nausea, vomiting, diarrhea, mild sialorrhea, symmetric mid-size pupils, wet skin, and bilateral moist rales in his lungs. His cardiac rhythm changed to rapid AF (HR >140 beats/*min*) after treatment with 3 *mg* midazolam followed by 0.3 *mg* of atropine (0.1 *mg* every 1-3 *min*). Atropine administration was discontinued and he was treated with 0.5 *mg* of digoxin. 6 *hr* later, his arrhythmia disappeared and all cardiac and laboratory evaluations changed to normal except reduced serum cholinesterase activity.

Conclusion: AF may be induced by Organophosphates (OP) and Pyrethroids (PYR) intoxication or during the treatment by atropine. We could not find any known risk factor (cardiac or medical issues) for AF in the current case. It may be suggested that poisoning with OP, PYR (alone or mixed) or atropine (in general or in low dose), or combination is the trigger of AF. However, AF is not life threating and can easily cure by antiarrhythmic therapy.

Keywords: Atrial fibrillation, Atropine, Cardiotoxicity, Organophosphates, Pyrethrins,

Introduction

Pesticide poisoning is one of the most common causes of hospitalization in the developing countries (1) as well as Iran. Pesticide poisoning is often caused by Organophosphates (OP), carbamate, and Pyrethroids (PYR).

OP and PYR affect many organs through different mechanisms. The main mechanism of OP is inhibition of acetylcholinesterase and increasing the concentration of acetylcholine in the biological environment (2,3) that lead to cholinergic and nicotinic toxidromes (2,3). PYR acts on voltage-sensitive sodium channels and delays the closure time (4). Therefore, PYR intoxication is manifested by nausea, vomiting, tremor, paraesthesia, hypersalivation, seizures, and depressed sensorium.

Although heart is not the main target organ of PYR or OP intoxication, it is affected in some cases (5). Some evidences have reported the ECG changes, and with lower incidence, Atrial Fibrillation (AF) in OP or PYR poisoning patients (4-10).

AF in OP poisoned patients had been reported in some other epidemiological studies, however, the information was ill-defined. Paul and Bhattacharyya (11) reported 5 cases (4.6%) of AF in 107 OP-intoxicated cases. Also, Makwana *et al* reported one case of AF between 50 OP-intoxicated cases, which was reverted by diltiazem injection (9). We report a rare case of OP+PYR poisoned patient who suffered from AF at the beginning of treatment by low dose of atropine.

Case Presentation

A 50-year-old man, following a family quarrel, ingested about 5-10 *ml* of a mixture of chlorpyrifos /cypermethrin near 16:30 pm at home. He had a history of lower limb partial paralysis due to spinal cord injury 10 years ago, without any past medical history of cardiac disease, hypertension, or diabetes. Half an hour after pesticide ingestion, he went to the rural hospital and complained of weakness. His vital signs were as follows: Hear Rate (HR)=105 beats/*min*, Blood Pressure (BP)=140/100 *mmHg*, Respiratory Rate (RR)=19 cycles/*min*, Temperature (T)=37.0°*C*, and Oxygen Saturation (O_2 sat)=89% (Table 1). Electrocardiogram (ECG) showed normal sinus tachycardia (Figure 1). He underwent gastric lavage and received 50 *g* activated charcoal. Then, he was treated by supportive care consisting of electrolyte solutions administration without any medication. After medical consultation with the regional clinical toxicology fellowship, the patient was referred to Korshid Hospital's clinical toxicology department affiliated with Isfahan University of Medical Sciences (CTD-IUMS).

The patient arrived in CTD-IUMS at 21:00 *pm* (4.5 hours after pesticide ingestion). On admission, he suffered from nausea, vomiting, diarrhea, and mild sialorrhea with no runny nose or lachrymation. He was lethargic [Glasgow coma scale (GCS)=14/15; E=4, V=4, and M=6], and had mild regular tachycardia (HR=90-110 beats/*min*) with BP=105/70 *mmHg*, T=37.1°*C* and RR=19 cycles/*min*. His weight was 75 *kg*, height was 183 *cm*, and BMI=22.4. On physical examination, he had symmetric mid-size pupils with normal response to light, wet skin, normal heart sounds, bilateral moist rales in lungs auscultation, and no specific smell. On ECG performed on admission in CTD-IUMS, he had normal sinus tachycardia.

Regarding some cholinergic manifestations (nausea, vomiting, diarrhea, mild sialorrhea, wet skin and bilateral moist rales in lungs auscultation), he was candidate to atropine therapy. After cardiac monitoring, the patient received slow infusion of 3 *mg* midazolam in 5 *min*, to control the tachycardia and reduce heart rate. His HR became less than 100 beats/*min* (Table 1). Then, he was treated with atropine. The first dose of Atropine (0.5 *mg*/*ml*) was diluted by 4 *ml* of distilled water and injected to the patient gradually, 1 *ml*/1-2 *min*. After administration of 3 *ml* (0.3 *mg* of atropine), his cardiac rate raised and became irregular (HR >140) and the distinct P wave disappeared on the bedside monitor. Other vital signs were included: GCS=13-14, BP=110/76 *mmHg*, and RR=18 cycles/*min*. The administration of atropine was held, and ECG was performed. Atrial fibrillation (AF) pattern with HR >140 was found in the ECG (Figure 1).

He was transferred to the Intensive Care Unit (ICU) at 00:00 *AM* and was treated with amiodarone 150 *mg* in 20 *min* continued by 360 *mg* for 6 hours, and 5000 units of low molecular weight heparin subcutaneously every 12 *hr*.

At 2:00 *am*, a cardiologist visited the patient

Table 1. The vital signs of organophosphate/pyretroid poisoned patient with atrial fibrillation

Day	Time	Event	HR	Regularity	SBP	DBP	RR	T	Sat	GCS
First day	17:00	Registration to the local hospital	105	Regular	140	100	20	37	89	14
	19:00	Discharge from the local hospital	85	Regular	125	75	18	37	95	15
	21:00	Registration to CTD-IUMS	110	Regular	105	70	19	37.1	92	14
	21:30	After Administration Midazolam	96	Regular	110	75	16	37	94	13
	22:00	After Administration Atropine	145	Irregular	112	79	18	37.1	94	13
	22:45	Registration to ICU	143	Irregular	118	82	17	37.1	94	13
Second day	00:00	Start amiodarone	146	Irregular	117	76	18	37.2	96	13
	1:30	-	140	Irregular	117	48	21	37.0	95	13
	2:00	Start digoxin	140	Irregular	120	67	20	37.0	97	13
	4:00	-	130	Irregular	120	58	21	37.0	97	13
	6:00	-	106	Regular	114	76	18	37.1	99	14
	7:30	-	108	Regular	106	82	15	37.5	99	15
	8:00	-	102	Regular	113	82	18	37.6	99	15
	10:00	-	92	Regular	109	70	19	37.5	95	15
	12:00	-	95	Regular	109	80	20	37.4	92	15
	14:00	-	93	Regular	132	88	23	37.3	95	15
	16:00	-	95	Regular	123	83	20	37.2	99	15
	18:00	-	87	Regular	120	89	21	37.0	96	15
	20:00	-	85	Regular	126	91	20	36.5	97	15
	22:00	-	89	Regular	120	88	21	36.6	96	15
	24:00	-	91	Regular	123	86	18	36.7	97	15
Third day	2:00	-	87	Regular	131	93	18	36.8	95	15
	4:00	-	91	Regular	125	89	19	36.7	96	15
	6:00	-	83	Regular	118	87	20	36.8	97	15
	7:30	-	91	Regular	118	82	21	-	-	-
	8:00	-	82	Regular	120	70	21	37	96	15
	10:00	-	83	Regular	96	56	20	36.8	94	15
	12:00	-	78	Regular	111	76	19	36.8	96	15
	14:00	-	75	Regular	121	80	18	36.5	94	15
	16:00	-	78	Regular	121	96	17	36.5	96	15
	16:30	-	96	Regular	129	85	17	-	93	-
	18:00	Retuned to ward	85	Regular	133	83	18	37	-	15
	21:00	-	108	Regular	122	78	20	36.8	-	15
	23:30	-	104	Regular	122	76	12	-	93	-
Fourth day	6:00	-	91	Regular	120	80	20	37	-	15
	8:00	Discharge	90	Regular	127	78	18	37.1	-	15

HR= Hear Rate (Beats/min), SBP= Systolic Blood Pressure (*mmHg*), DBP= Diastolic Blood Pressure (*mmHg*), RR= Respiratory Rate (Cycles/min), T= Temperature (°C), O2 Sat = Oxygen Saturation (%), GCS = Glasgow Coma Scale.

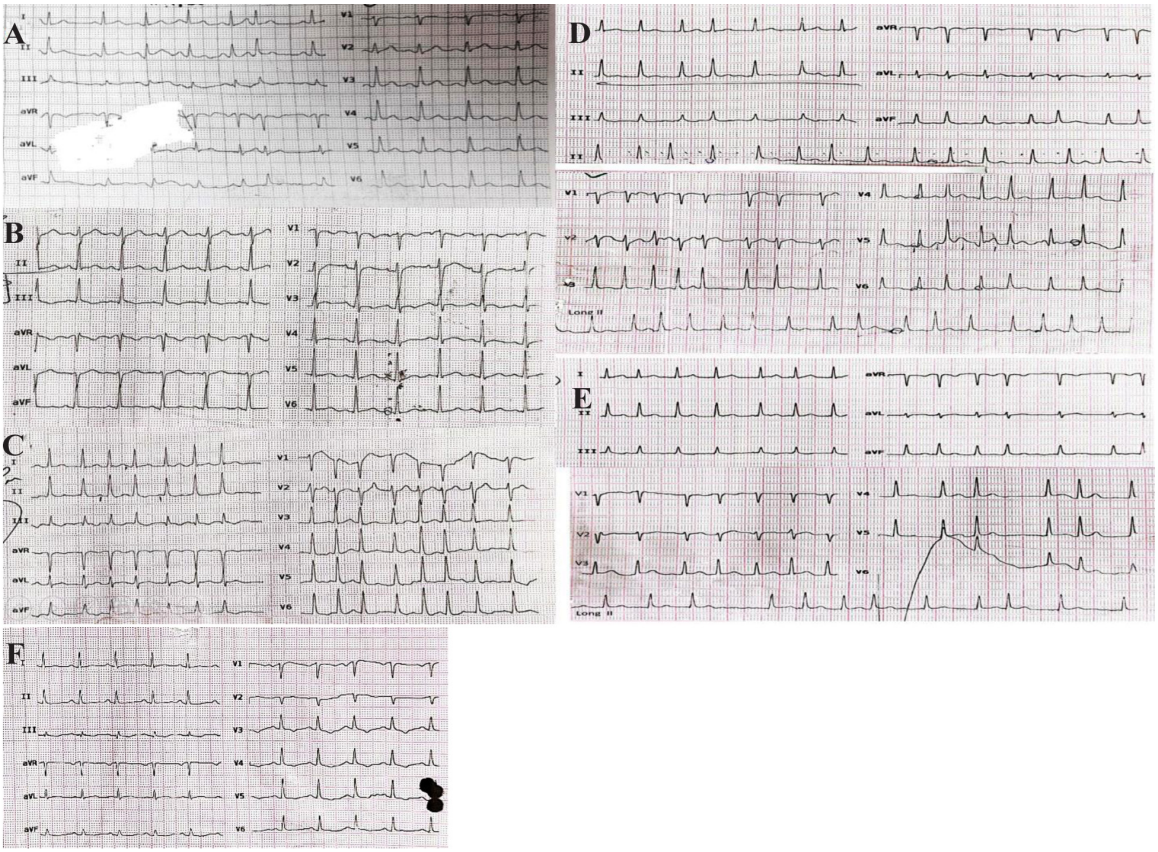

Figure 1. The electrocardiogram of organophosphate/pyretroid poisoned patient with atrial fibrillation.
A=At 17:00. On the first day, on registration to the local hospital. B= At 21:00. On the first day, on regis-tration to CTD-IUMS. C= At 21:30. On the first day, after atropine administration, atrial fibrillation. D= At 1:32, On the second day, ICU admission, atrial fibrillation.E= At 2:30, On the second day, after receiving loading dose of amiodarone, atrial fibrillation. F= At 6:00, On the second day, normal rhythm.

and reported the irregular HR >140 beats/*min*, BP=121/91 *mmHg*, O2 sat=93%, reduced first heart sound, and moist rales in both lungs. His bedside echocardiographic assessment findings were as: Left Ventricular Ejection Fraction (LVEF)=35-40%, no pericardial effusion, no systolic, and no valvular dysfunction. However, concerning his tachycardia echocardiographic, the findings were not accurate. Regarding the recommendation of the cardiologist, amiodarone was discontinued and 0.5 *mg* of digoxin was injected. He also received 40 *mg* of Furosemide which repeated 20 *mg* every 8 hours. About one hour later, the patient's HR decreased (HR=130) (Table 1) and two hours later, the patient's pulmonary crackles decreased. At 6:00 *am*, the patient had a normal sinus rhythm (HR=106) and no abnormal sound was auscultated on his lungs.

On the second day, he underwent echocardiography again. Echocardiographer reported: normal left ventricular size, LVEF=50-55%, normal systolic function, grade one of diastolic dysfunction, no prominent valvular abnormality and normal Pulmonary Artery Pressure (PAP=30 *mmHg*).

He received no atropine or pralidoxime during hospitalization. He was treated by oral hyoscine tablet 10 *mg* three times per day for his mild cholinergic manifestations. All laboratory findings, including troponin, were normal, except cholinesterase serum activity (Table 2). On the third day, he was returned to the clinical toxicology ward, and after 12 hours, he was discharged in good condition and suggested to visit the psychiatrist.

Discussion

We reported a rare case of AF after ingestion of 5-10 *ml* chlorpyrifos/cypermethrin mixture. Patient's cardiac rhythm changed to rapid AF (HR >140 beats/*min*) after treatment with low dose of atropine (0.3

Table 2. The laboratory findings of organophosphate/pyretroid poisoned patient with atrial fibrillation

Test (unit)	Day →	1th		2nd		3rd	
	Time →	21:10	2:55	04:30	10:59	4:0	5:54
pH		7.297	-	7.355	-	-	-
PCO_2 (*mmHg*)		47.1	-	52.2	-	-	-
HCO_3 (*mEq/L*)		22.3	-	28.4	-	-	-
Base excess		-4.3	-	1.7	-	-	-
With blood cell count (*1000/*ml*)		11.4	-	10.1	-	5.3	-
Neutrophils (%)		93.4	-	75.1	-	59.0	-
Lymphocytes (%)		4.5	-	13.0	-	28.1	-
Red blood cell count (*10^{12}/*µl*)		4.33	-	4.28	-	4.03	-
Hemoglobin (*g/dl*)		17.0	-	17.2	-	16.2	-
Hematocrit (%)		48.1	-	47.3	-	44.8	-
Platelet count (*1000/*ml*)		196	-	232	-	247	-
Partial Thromboplastin Time (*S*)		27	-	27.7	-	28.6	-
Prothrombin Time (*S*)		14.2	-	14.4	-	16.1	-
International normalized ratio		1.11	-	1.13	-	1.29	-
Blood Urea Nitrogen (*mg/dL*)		17.6	-	16	-	10	-
Creatinine (*mg/dL*)		0.87	-	0.90		0.99	-
Aspartate transaminase (*U/L*)		18	-	24	-	17	-
Alanine transaminase (*U/L*)		17	-	17	-	14	-
Alkaline phosphatase(*U/L*)		398	-	367	-	302	-
Sodium (*mEq/L*)		141	-	142	-	141.5	-
Potassium (*mEq/L*)		4.1	-	4.6	-	3	-
Blood sugar (*mg/dL*)		136	-	147	-	89	-
Troponin I (*ng/ml*)		-	11.7	-	14.6	-	-
Serum cholinesterase (*U/mL*)		264	-	-	-	-	320
Creatine phosphokinase (*IU/L*)		64	-	-	-	-	-
Lactate Dehydrogenase (*IU/L*)		312	-	-	-	-	-
Triiodothyronine (*ng/dL*)		-	-	-	-	70	-
Thyroid stimulating hormone (*IU/L*)		-	-	-	-	2.5	

mg atropine, 0.1 *mg* every 1-3 *min*). No underling cardiac problem was found. The patient was treated by digoxin and discharged in a good condition.

AF is rarely reported in OP or PYR-poisoned patients; however, the studies review and describe the cardiac manifestation of OP or PYR poisoning are not rare. The literature was searched and seven cases of pesticide-induced AF were found (Table 3). Four cases were intoxicated by OP (the exact agent of two cases were not defined), two cases by carbamate, and one case by PYR (cypermethrin). In line with our report, cypermethrin and chlorpyrifos are two agents

Table 3. The summary of organophosphates and/or pyrethroids induced atrial fibrillation cases

Authors (y) (ref)	Age (Y)/ sex	Agent/ rout/ Couse	Vital sign	CTA	Signs and symptoms	ECG	Cardiac en-zymes	Lab tests	Cardiac evaluation	General treatments	Anti-arrhyth-mia	Duration of AF (H)	LOS
Ustundag *et al* (12)	25/M	Cyper-methrin/ Inh/ACS	BP= 120/ 80 *mmHg*	2	Nausea, vomiting, dizziness, palpitation, paresthesia	AF HR= 115	-	Normal	Normal TTE	supplementary treatment	NO	12	24 *hr*
Ustundag *et al* (12)	30/M	Dic-hlorvos/ Ing/ACS	BP= 130/ 80 *mmHg*	1	Hypersaliv-ation, nausea, vomiting. No hypoxia	AF HR= 120	Normal	Normal	Normal TTE	Gastric lavage, Active charcoal, pralidoxim =1 *g*	NO	6	72 *hr*
Topacoglu *et al* (19)	26/M	working in a carbamate production factory	BP= 109/ 66 *mmHg* HR= 64 RR= 20 Sat= 97	1	Nausea, Vomiting, Myosis, Dyspnea. No hyperse-cretion	AF Normal ventri-cular response, no ST/T changes.	Normal	Normal, ACE =mild reduced	Normal TTE	External detoxification, supplementary treatment, acetylsalicylic acid=300 *mg*	NO	24	-
Abdelnaby (7)	12/M	OP/?/ ACS	BP= 100/60 *mmHg* PR= 120		Miosis, Fascicul-ation, crackles in both lungs.	AF Rapid ventri-cular response.	Normal	Normal	Normal TTE	Atropine, Pralidoxime	direct current cardio-version.	-	48 *hr*
Patel *et al* (8)	42/M .	250 *ml* of chlor-pyrifos/ Ing/?	BP= 150/90 *mmHg* PR= 140 RR=28 Sat= 98	3	Dyspnea Altered behavior, Sialorrhea, Vomiting, Crackles in both lungs. Sweating, Garlic odour smell,	AF Rapid ventri-cular response.	-	Normal	Normal TTE	Atropinizatio, pralidoxim =2 *g*	metoprolol = 5 *mg*. (Not response) then Amiodarone = 150*mg* - 60*mg*/*kg* -30*mg*/*kg*	16	6 days
Maheshwari & Chaudhary (6)	40/M	OP/Ing/ ACS	BP= 110/80 *mmHg* HR= 70		Miosis, Crackles in both lungs. Fascicul-ation,	AF	-	Normal	Normal TTE	Atropine, Pralidoxime, Magnesium, sulfate	NO	-	-
Mazaraki *et al* (20)	50/M	Lannate /skin contact/ ACS	BP= 120/80 *mmHg* HR= 145	2	Chest discomfort, Palpit-ations, Dizziness, Mild itching	AF Rapid ventric-ular response. 0.5 *mm* horizontal ST-depres-sion in leads V4–V6.	Normal	Eosino-phils = 800 cells/*µl*	Normal coronary angiogram	enoxaparin 1 *mg*/*kg*	Esmolol = 50 *µg/kg/*	6	3 days

TTE= Trans thoracic echocardiography, ACS=Accidentally, SA= Suicidal attempt Ing=Ingested Inh= Inhaled, ACE= acetylcholine esterase
BP= Blood pressure (mmHg), HR= Heart Rate (beats/min), RR= respiratory rate (Cycle/min), Sat= Oxygen saturation (%), CTA= Lag Time between contact to admission (hours), ECG= Electrocardiogram findings, AF= Atrial Fibrillation, LOS= Length Stay in hospital.

that induced AF in reported cases (8,12). Pesticide intoxication was the only reported risk factor for AF and all the other laboratory and cardiac evaluations were unremarkable. Three cases received specific antiarrhythmic therapy. And all of them returned to normal sinus rhythm 6 to 24 *hr* later. In contrast to the current case, all cases had AF before starting the treatment, while in the present case, AF was triggered by atropine (Table 3). It seems that there is no difference between the AF rhythm lasting on treated and non-treated patients.

AF is the most common arrhythmia; however, it is rare to happen in pesticide intoxication. Both tachycardia (as in the case of PYR poisoning or during atropinization) and bradycardia (as OP poisoning) could trigger AF (13). In patients who are susceptible to atrioventricular nodal reentrant tachycardia (AVNRT) and atrioventricular reentrant tachycardia (AVRT), AF is triggered by tachycardia (13). Bradycardia that is induced by vagus nerve stimulation, such as during sleep, also mediates AF (13).

AF is facilitated when action potential duration is shorted by reducing the wavelength for reentry. Action potential duration in atrial cells is shorted by acetylcholine. OP increases acetylcholine levels through inhibition of acetylcholinesterase enzyme (2). American Heart Association reported that Physostigmine, a carbamate, as a acetylcholinesterase inhibitor, mediated AF by increasing the level of acetylcholine in the heart (14). Saito *et al*, also evaluated the incidence of AF in patients undergone intracoronary acetylcholine provocation test for assessment of vasospastic angina. They found that about 8% of the patients who received acetylcholine injection into the coronary arteries showed AF (15). It is similar to incidence of AF among OP poisoned patients reported by Paul and Bhattacharyya (4.8%) (11). Saito *et al* also reported that patients with lower body mass index (BMI) are at higher risk of AF in response to acetylcholine (15) and it is much rare in obese patients. Our case had normal BMI (22.4).

Overactivity and dysfunction of sodium channels of cardiac cell are suggested as main mechanisms of AF. However, cypermethrin is not commonly cardiotoxic; the results of animal models have been shown histologic, functional, and ECG abnormalities (4) and therefore, it can be considered as a cardiac arrhythmogenic in mammal. Cypermethrin reduced the amplitude and duration of the P wave, atrial muscles contraction forces, and induced cardiomyopathy in frog (10). Cypermethrin, as a PYR, delays the closure of voltage-sensitive sodium channels (4). The cardiac myocytes are also rich in voltage-sensitive sodium channels. Thus, cypermethrin could lengthen the duration of sodium channel opening, prolong cardiac action potential, and evoke afterdepolarizations in myocytes resulting in an increase in the variability of contractile force and potential arrhythmias.

The current case ingested a combination of chlorpyrifos and cypermethrin pesticides and AF was reported by both of them (8, 12). Iyyadurai *et al* compared the patient poisoned by chlorpyrifos, cypermethrin, or combination of two pesticides and revealed their combination can induce more severe poisoning and worse outcomes (16).

Atropine is an antagonism of muscarinic receptors that enhances sinus node automaticity and atrioventricular conduction resulting in tachycardia and maybe AF. However, the sensitivity of atropine in the different structure of heart is various and this variation could lead to unpredictable and apparently paradoxical effects. Moreover, AF had been reported in patients who received atropine for relieving non-OP induced bradycardia (17). Although atropine is the main antidote of OP poisoning and the patients sometimes received a large dose of atropine for a long time (2), AF is rare in OP poisoned patients. Three of forth OP-induced AF cases reported, continues high doses of atropine administration even after AF occurrence (Table 3). Their cardiac rhythm spontaneously returned to normal (6-8). However, in the current case, atropine administration was discontinued when AF presented and no more atropine was needed to control the symptoms. The current patient developed AF after receiving low dose of atropine (about 0.3 *mg*). Atropine produces a biphasic effect on cardiac rate. Low dose of atropine induces bradycardia and continuous infusion of low dose atropine (about 0.12 *mg*) augments the parasympatomimetic effects and reduces the heart rate and raises beat-to-beat heart rate variability. The low dose atropine-induced sinus bradycardia is mediated by a central action, cholinesterase inhibition and enhancement of vagal

outflow to the heart. The following hypothesis is suggested by authors: the low dose administering of atropine in the current case caused more acetylcholinesterase inhibition, which was previously inhibited by organophosphorus, and therefore might increase the amount of acetylcholine in the heart that can cause AF (15).

It is also suggested that hypoxia may be a cause of arrhythmia in OP poisoned patients during atropine administration. Hence, oxygen therapy is recommended before atropine administration (2). The result of a cohort study showed that the hypoxia did not increase the risk of cardiac arrhythmia in OP poisoned patients (18). However, the present case was not hypoxic at any time before and during the atropine administration (Table 1). Also, none of the reviewed cases (Table 3) had hypoxia or reduced oxygen saturation. It seems that hypoxia may not be a trigger or risk factor for the patient with dysrhythmia.

We found no known risk factor (cardiac or medical issues) associated with AF in the current case. It may be suggested that poisoning with OP, PYR (alone or mixed), atropine (in general or in low dose), or a combination of them is the trigger of AF occurrence in the patient.

The pesticides' serum levels were not evaluated in the present study. Also, we had to rely on the patient's self-expression about past history of heart disease.

In conclusion, AF may occur by OP and PYR intoxication or during the treatment by atropine. It is not life-threating and is self-limited or is easily cured by routine antiarrhythmic therapy.

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Conflict of Interest

All authors declare no financial/commercial conflicts of interest.

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