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CASE REPORT

Alexandria Poison Center Case Report of Acute Poisoning Following Intramuscular Injection of Cholinesterase Inhibitor Insecticide and the Possible Role of L-Carnitine in Management

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Abstract

Background: Cholinesterase inhibitor (ChEI) insecticide is a common cause of poisoning in developing countries. Toxicity occurs by ingestion, inhalation, or dermal absorption. However, only a few cases of parenteral poisoning have been reported, so far. The inhibition of cholinesterase itself is not enough to explain the wide range of disorders associated with ChEIs insecticide exposure. Oxidative stress is supposed to be a contributing factor to the complications of ChEIs insecticide poisoning. L-carnitine (LC) is a widely accessible antioxidant therapy that is a safe drug with fewer side effects and it could be considered as a promising adjuvant treatment in acute ChEIs insecticide poisoning.

Case Presentation: The current case report describes a 60-year-old male with a homicidal intramuscular injection of ChEI insecticide, presented with chest crepitations and fasciculations. He showed an initial improvement to treatment with atropine, toxogonin, and L-carnitine (LC) and discharged. However, there was an aggressive reaplse of symptoms after five days necessitating readmission and mechanical ventilation. After two weeks of treatment with the same regimens, the patient's condition improved significantly and he was discharged with complete recovery.

Conclusion: The diagnosis of ChEI toxicity by the parenteral route is a challenge, where the onset of symptoms may be delayed with atypical presentations. Even though the symptoms are mild initially, observation for a more extended period is mandatory. LC could be a promising adjuvant antioxidant treatment in acute ChEIs insecticide poisoning cases.

Keywords: Homicidal; Cholinesterase Inhibitor Insecticide; L-carnitine; Case Report.

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INTRODUCTION

Insecticide poisoning is a significant problem worldwide, especially in developing countries. Egypt as one of the most populated countries in Africa relies primarily on agriculture, as one of its main sources of national income. As a result, Egypt consumes great amounts of pesticides to control pests [1, 2].

The most extensively utilized types of insecticides are cholinesterase inhibitors (ChEIs), predominantly organophosphorus (OP) compounds, and carbamates. They exert their toxicity mainly by inhibiting cholinesterase Enzyme (ChE) in the nerves and muscles [3].

Poisoning occurs mainly by ingestion, inhalation, or absorption through the skin. However, only a few cases of parenteral poisoning have been reported. Most cholinesterase inhibitors are lipophilic and are therefore predicted to have a large volume of distribution and to distribute into tissue and stored mainly in fatty tissues rapidly [4].

The main presentation of (ChEIs) is acute cholinergic crisis. Respiratory failure is frequently implicated in mortality. However, intermediate syndrome persists to be a problem causing a significant burden on health care facilities [5, 6].

The mainstays of treatment for acute ChEIs insecticide poisoning are supportive care, oxygen, atropine, and oximes. Although the current treatment strategy is effective, the rate of case fatalities is still high [7].

It is also argued that the inhibition of cholinesterase itself is not enough to explain the wide range of disorders associated with ChEIs insecticide exposure. According to some researchers, oxidative stress is the mechanism underlying the complications of ChEIs insecticide poisoning [8].

L-carnitine (LC) is a widely accessible antioxidant therapy that is a reasonably priced, safe drug with fewer side effects. It is extensively as a marvelous treatment in many health conditions. L-carnitine has an immunological role in diabetic individuals. Also, it has shown potential for treating several neurological diseases [9].

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Recently, LC has attracted increasing concern as a treatment for various xenobiotics-induced toxicities, where oxidative stress is believed to be a contributing factor. It can encourage the long-chain fatty acids β-oxidation, plays a role in the metabolism of branched-chain amino acids, and maintains the integrity of cell membranes. Additionally, it can improve the human antioxidant enzyme activities. L-carnitine protects antioxidant enzymes from oxidative damage and modulates the activity of enzymes involved in defense against it [10].

This case study discusses a case of homicidal intramuscular ChEIs insecticide poisoning. Interestingly, the onset of symptoms was about 4 hours after injection and there was a relapse of severe manifestations after the first discharge.

This case report was conducted to illustrate the hazards of parenteral injection of ChEIs insecticide together with studying the possible role of L-carnitine in the management of acute ChEIs insecticide poisoning.

CASE PRESENTATION

A 60-year-old male patient has been admitted to the Alexandria Poison Center (APC) with an allegation that someone injected him with an unknown substance. The injection site was observed in the left subscapular region it was painful and edematous (Fig 1). Four hours after the incident, he presented with spontaneous fasciculations (facial and in both lower limbs). The past medical history was irrelevant. The patient did not seek medical advice before being admitted to APC.

Upon examination, the patient was fully conscious. Vital signs revealed a pulse rate of 100/minute, blood pressure of 130/80 mmHg, respiratory rate of 18 cycles per minute, a temperature of 36.9^0 C, and pulse oximeter showing 90% saturation on room air. The electrocardiogram (ECG) was normal with a corrected QT interval (QTC) of 0.50 seconds.

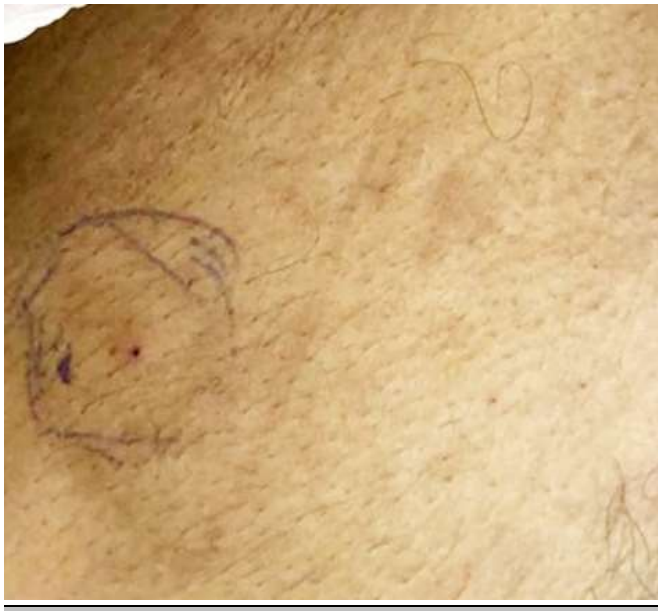

Figure 1. The injection site

Neurological examination revealed normal cranial nerves. Miosis was detected bilaterally (pinpoint pupils (ppp)) with no reaction to light. Chest examination showed diminished air entry bilaterally with heard coarse crepitations.

The examination of other systems was normal. Blood gasses analysis showed average values. Chest X-ray showed increased broncho vascular markings. Regarding laboratory investigations, CBC showed leukocytosis with a white blood cell count (WBC) of $23.3x10^3/\mu L$ (normal range: 4-11 $x10^3$ /μL). Cardiac enzymes showed increased creatine kinase (CK total was 300 U/L, normal range: 0-195 U/L). The plasma cholinesterase level was 1437 U/L (normal range: 5600-11200 U/L). Oxidative stress biomarkers were measured and showed serum MDA = 6.9 nm/ml and GSH = 1.53 mg/dl. Other laboratory findings were within average values.

The diagnosis of the case as acute ChEI poisoning was based mainly on the characteristic cholinergic toxidrome with its full-blown picture of muscarinic and nicotinic manifestations. Also, the severely decreased cholinesterase level and the rapid response to the atropine trial confirmed the provisional diagnosis.

The main differential diagnosis of the presenting symptoms was acute opioid toxicity which was excluded by negative drug in the urine screening test. The patient was treated with atropine, toxogonin, and L-carnitine. Atropine was given intravenously as a bolus dosage of 1 to 3 mg IV, repeated every 10 to 15 minutes until clearing of respiratory secretions (seven ampoules), and then one ampoule continued as a maintenance dose (10–20% of the loading dose every hour). Toxogonin® (1 ampoule containing 250 mg of obidoxime chloride in 1 ml) was administrated at 250 milligrams (mg) as an intravenous bolus, followed by a continuous infusion of 750 mg over 24 hours diluted in 500 ml normal saline at a rate of 20ml/hour. L-carnitine was given by intravenous infusion as a 100 mg/kg loading dose and 50 mg/kg maintenance dose every 8 hours (a total of 10 ampoules during 24 hours).

The patient showed an initial improvement to atropine, toxogonine, and L-carnitine treatment strategy which was continued for 24 hours. After treatment, the serum cholinesterase enzyme (AChE) was 1878.7 U/L, MDA 9.6 nm/ml, and GSH 1.73 mg/dl. The patient was discharged from the hospital after three days with complete recovery.

Five days later, the patient was readmitted to APC with salivation, dyspnea, and bilateral spontaneous lower limb and facial fasciculations. The patient was drowsy with constricted, sluggishly reactive pupils. Chest examination showed decreased air entry bilaterally with coarse crepitations heard on auscultation that were cleared after atropine therapy (15 ampoules). Oxygen saturation was 89% on room air and 99% on the O_2 mask, and serum cholinesterase was 1484.7 U/L.

The treatment protocol with atropine, toxogonin, and L-carnitine was re-administrated with the same regimen. After two days, the condition deteriorated and the patient developed bronchorrhea, fever with a temperature of 39.5, hypoxia with an O_2 saturation of 81% on room air, and proximal muscle weakness. The patient received 76 atropine ampoules (63

ampoules loading and 13 ampoules as maintenance dose) and was then transferred to the ICU, where he was intubated and mechanically ventilated for 24 hours. During ICU admission, sinus tachycardia was monitored with a heart rate of 105 beats/minute.

The patient continued on the same maintenance doses of toxogonin and L-carnitine for seven days. The general condition was stable, but he was sleepy in the first four days. Antibiotic therapy with meropenem was started with a dose of 1 gram every 8 hours. Starting from the fourth day, the dose of L-carnitine was doubled. On the sixth day of ICU admission, L-carnitine continued alone without atropine or oximes after apparent recovery from the muscarinic and nicotinic manifestations. Also, the level of consciousness was improved with a GCS =15.

The patient was discharged from the ICU with a 1304 /L cholinesterase level. After discharge to the ward, the patient complained of severe weakness and inability to raise his head or use the proximal limbs. The muscle power was 3/5, which raised suspicion about the intermediate syndrome (IMS), but the cranial nerve functions were normal. Treatment with toxogonin and l-carnitine was resumed and continued for three days.

Adherence and tolerability to interventions were assessed by monitoring the patient's clinical condition and assessment of lab investigations. Also, an electrophysiological study of both upper and lower limbs was conducted and revealed evidence of myopathy, most probably critical illness myopathy (Fig 2). Repetitive nerve stimulation of both upper and lower limbs was normal proximally and distally.

Finally, the patient was able to walk without support before being discharged from the hospital on day 15. Upon discharge, the cholinesterase level was 1337 U/L. In this case of acute parenteral cholinesterase inhibitor (ChEI) poisoning, several hazards and complications were observed including initial cholinergic toxidrome and respiratory compromission leading to hypoxia and the need for mechanical ventilation. Cardiac involvement was also manifested by prolonged QTC upon presentation and sinus tachycardia during ICU admission. Muscle weakness, particularly in proximal limbs, possibly resulting from prolonged ChEI exposure was another critical clinical finding.

The utilization of L-carnitine treatment in this scenario could elucidate the relatively rapid recovery of the patient, the reduced necessity for atropine and oximes, and the rapid weaning from mechanical ventilation. Furthermore, it may have contributed to the patient's survival and acted as a safeguard against the emergence of a complete intermediate syndrome (IMS) manifestation.

DISCUSSION

Poisoning by ChEI insecticides via ingestion, inhalation, or dermal contact is common. The use of the parenteral route to administer this poison is extremely rare. Parenteral injection could be suicidal or homicidal. Also, it could be intramuscular, intravenous, or subcutaneous [11-13]. In cases of suicidal injection of organophosphate (OP), the most common site for injection was the upper limb, followed by

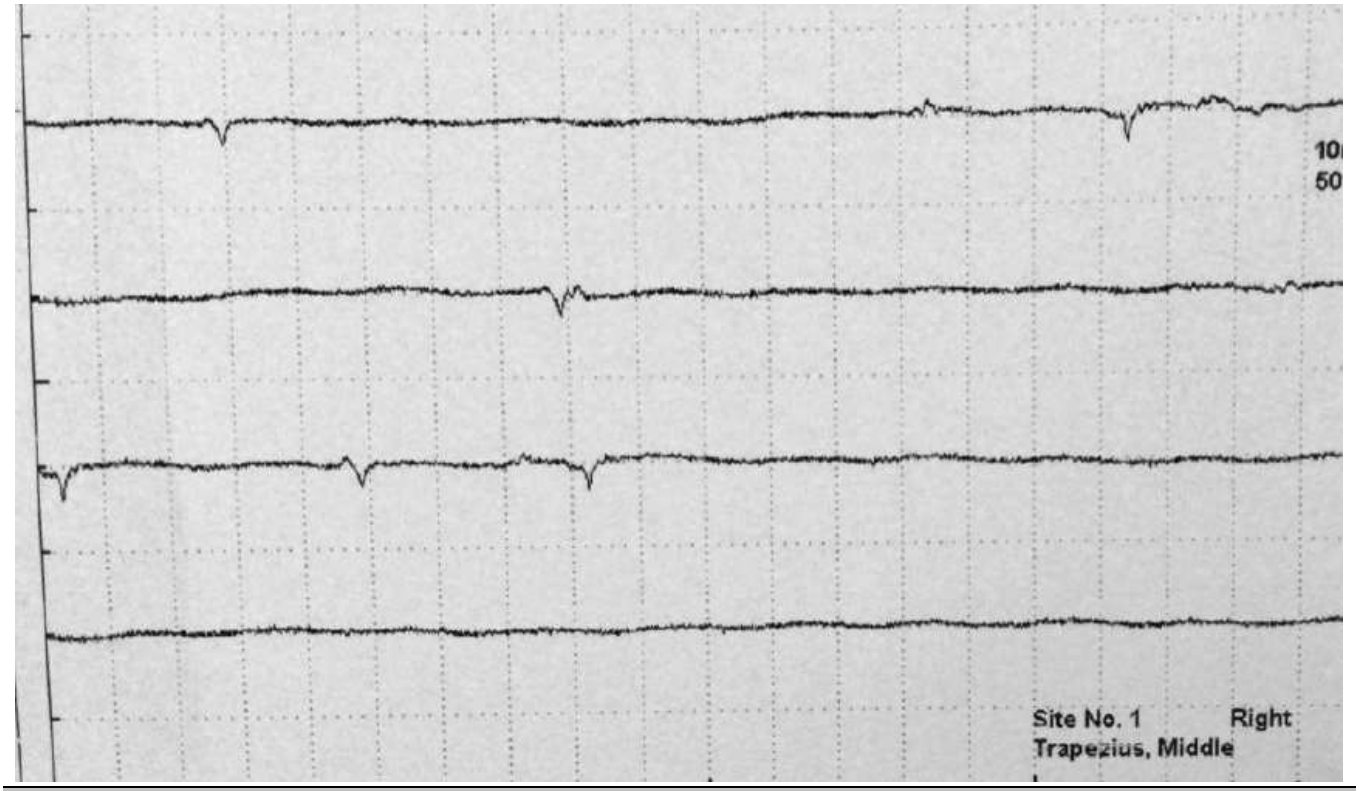

Figure 2. Trapezius muscle electrophysiology showed normal motor amplitude and no decrement on repetitive nerve stimulation

the abdomen. One case of suicidal injection reported the calf muscle as a site of injection. Also, one case reported intravenous self-injection [12, 14]. The back was the injection site in this studied case with a clear needle mark about 1 ml in diameter. This inaccessible area of the body excludes suicidal attempts and is highly suggestive of homicide. In 2012, Kumar et al [15] reported a case of homicidal insecticide poisoning by intraperitoneal injection.

Literature review in the PubMed, ISI, and Embase databases reported the onset of symptoms of injected organophosphate compound poisoning to range from 30 minutes to four hours. In the present study, the onset of symptoms was about 4 hours. In 2013, Liu et al. reported a case of suicidal IM injection of insecticide that had occurred nine days before admission [11]. In cases of parenteral injection of ChEI, attention must be paid to the possibility of late-onset clinical manifestations due to the slow rate of absorption that is affected by many factors. Most of cholinesterase inhibitors are lipophilic and are therefore predicted to have a large volume of distribution and to rapidly distribute into tissue and stored mainly in fat reservoirs [16] .

Also, the chemical nature of the ChEI compound may affect the onset of symptoms. Some pesticides require partial metabolic activation in the intestinal mucosa and liver to more active forms that inhibit cholinesterase enzyme directly [16]. Consequently, pharmacokinetics and dynamics properties including the degree of lipid solubility of the injected drug, local blood flow, the total surface area of muscle injected, muscle activity, and subcutaneous fat thickness may affect the onset of presentations [17, 18]. Moreover, the severity of symptoms depends also on the percentage and amount of poison consumed [19].

Local complications at the site of the injection are expected findings in the case of parenteral poisoning, mainly due to the vehicle solvent of the ChEI or the use of contaminated syringes. Consequently, tetanus prophylaxis and antibiotic therapy are mandatory [20]. In the presented case, the injection site was only painful and edematous without any local complications. Similarly, Erenler et al [21] reported a case of suicidal injection of an insecticide in the upper limb with erythema and swelling. However, Agrawal et al [22] and Liu et al [11] reported cases of suicidal intramuscular injection of insecticides with cellulitis and abscess formation that required surgical debridement .

One of the most serious complications of ChEI insecticide poisoning is cardiac toxicity including electrocardiogram changes. The ECG findings of the current case showed a prolonged QTC interval. The prolonged QT interval originates from intense and unequal sympathetic stimulation of myocardial fibers. Therefore, both sympathetic and parasympathetic stimulation may cause QT-interval prolongation [23]. Khan et al reported a case of organophosphate poisoning with other ECG changes including a biphasic T wave in leads V2, V3, V4, and V5[24] .

The laboratory investigations of the current case showed elevated creatine kinase (CK) levels. The same finding was reported in another case of parenteral OP poisoning [25]. There are several studies demonstrating the role of CK as a prognostic biomarker in cases of acute ChEI insecticide poisoning [26-28]. This case demonstrated the seriousness of early discharge of patients with parenteral ChEI poisoning. The relapse with more aggressive manifestations occurred after five days of complete recovery and discontinuation of treatment. This could be explained by the slow release of the poison from the fat reservoir and muscular depot at the site of injection. Nizami et al [29] reported a case that also showed deterioration of the patient's condition after seven days of admission.

The development of respiratory failure and the need for mechanical ventilation are serious complications of ChEI poisoning. Here, the studied case was mechanically ventilated after two days of readmission but only for twenty-four hours. Other cases, such as those reported by Juluganti et al [30] and Singh. et al [31], required ventilator support for twelve and eighteen days respectively. In the present case, the patient received eight atropine ampoules in the first admission and seventysix ampoules in the second one. Agrawal et al [22] reported a case of intramuscular OP injection that received 160 atropine ampoules. Decreasing the dose of atropine is an important clinical concern because its administration in high dosages is usually accompanied by serious complications. The lower doses of atropine used in the current case may be attributed to the concurrent use of L-carnitine and its antioxidant effect in cases of acute ChEI insecticide poisoning [32] .

The development of sinus tachycardia during ICU admission may be attributed mainly to atropine therapy as an anticholinergic agent. Tachycardia is a common cardiotoxic effect of atropine due to its chronotropic effect on the heart. It also causes arrhythmias such as atrial fibrillation (AF), atrioventricular dissociation, ventricular fibrillation, and ventricular tachycardia. Chakravarthula et al, reported a case of OP poisoning complicated with AF as an adverse effect of atropine treatment of OP poisoning [33]. Regarding the correlation between plasma cholinesterase enzyme level and the patient's clinical presentation after parenteral toxicity, the ChE level was 1437 and 1484 U/L in the first and second admissions, respectively. Upon discharge, the ChE level was 1337 U/L. In another case of intramuscular injection of CEI insecticide reported by Liu et al, serum cholinesterase level was 200 IU/l on admission and increased to 3,823 IU/l after twenty-one days of hospital admission [11].

In patients with ChEI intoxication by parenteral route, the serum cholinesterase level of the patient has a disparity with the clinical severity of intoxication. Considering this disparity, clinical severity should be considered the most significant indicator for the clinical assessment and treatment of OP poisoning rather than cholinesterase level. Also, the enzyme may take about 4-5 weeks to regain its normal level [34]. ChEIs can produce free radicals and change antioxidant capacity by inducing oxidative stress, which leads to scavenging antioxidant enzymes [35] .

Early treatment with anticholinergic drugs, such as atropine and oximes, that reverse the AChE inhibition is the primary intervention to prevent acute symptoms of toxicity. Therefore, drugs with antioxidant effects might be a promising therapeutic option to dampen the increased reactive oxygen species (ROS) and replenish the antioxidant enzyme system [36]. Despite the aggressive relapse of presentations in the second admission, the total duration of

hospital stay was only fifteen days and the patient was discharged with complete recovery. Repetitive nerve stimulation (RNS) revealed normal nerve conduction in both upper and lower limbs, and electromyography explained that the developed myopathy was most probably due to critical illness. Nizami et al reported a case of intramuscular OP poisoning that was complicated with intermediate syndrome and discharged after twenty-two days of admission [29] .

Table (2) provides a comparative overview of cases of intramuscular ChEI insecticide poisoning presented with acute cholinergic toxidrome. Although the levels of oxidative stress biomarkers did not change significantly in the current case, the role of L-carnitine was observed clinically. The use of L-carnitine therapy, in this case, may explain the relatively rapid improvement of the case, the lower required doses of atropine and oximes compared with other studies, and the rapid weaning from mechanical ventilation and contribute to the survival of the case and protect against the occurrence of a full-blown picture of the intermediate syndrome (IMS) [31, 37].

LIMITATION

Unavailability of the injected substance to be analyzed and the lack of the exact dose injected to assess the case effectively. Another limitation is the early discharge of the patient in the first admission, as he should have been kept under observation for a longer time.

CONCLUSION

ChEIs toxicity by parenteral route is a diagnostic challenge. The onset of symptoms may be delayed with atypical presentations. Even though the symptoms are mild

Table 1. Laboratory results during the hospital stay.

Test		1st admission	2nd admission	
Hematology	Hb (g/dl)	14.5		13-17
	Hct (%)	42.7		40-50
	RBCs ($x10^6$ /µL)	4.5		4.5-5.56
	WBCs ($x10^3$/µL)	23.3		4-11
	Platelets ($x10^3$ /µL)	350		150-400
	Eosinophils ($x10^3$ /µL)	0.3		0.0.6
	Pt (sec)	11.8		11-14
	Prothrombin Activity (%)	95.6		70-130
	INR	1.03		0-1.2
Electrolytes	Na+ (mmol/L)	138	141	136-145
	K+ (mmol/L)	3.7	3.8	3.5-5.1
Liver profile	AST (U/L)	19		15-37
	ALT (U/L)	25		16-63
Renal profile	Urea (mg/dl)	32	45	15-45
	Creatinine (mg/dl)	0.9	0.9	0.7-1.3
	BUN (mg/dl)	15	21	7-18
Cardiac enzymes	CK-Total (U/L)	300		0-195
	CK-MB (ng/ml)	0.0	3.5	0-5
	Troponin](ng/L)	0.00	0.01	0-0.05
ABG	pH	7.45	7.38	7.35–7.45
	pCO2 - (mmHg)	21	44	35-45
	pO2 - (mmHg)	173	75	80-105
	HCO3 − (mmol/L)	14.6	26	22-28
	SO2 − (%)	100	96	>95
Cholinesterase enzyme (AChE) U/L	Before treatment	1437	1484.7	5600-11200
	After treatment	1878.7	1337	
Oxidative stress markers	MDA (nm/ml)	Before treatment:6.9		
		After treatment : 9.6		
	GSH (mg/dl)	Before treatment:1.53		
		After treatmet :1.73		

Table 2. A comparative overview of cases of intramuscular ChEI insecticide poisoning with varying outcomes and treatment durations.

The study	circumstances	Area of injection	Time before admission	Need for mechanical ventilation	Duration of antidote therapy (atropine&oximes)	Complications with intermediate syndrome	Duration of hospitalization	Outcome
Agrwal et al. 2015[22]	case1: Suicide case 2: suicide	Case 1: Arm (Deltoid muscle) Case 2: Arm (Deltoid muscle)	_____	no	Case 1:160 atropine ampoules over 15 days Case 2 :80 atropine ampoules over 14 days	no	Case 1 : 15 days Case 2: 14 days	recovery
Premaratna et al. 2001[14]	Suicide	Leg (Calf muscle)		Yes (10 days	١٤ days	no	_____	recovery
Nishioka Sde A. 1994[38]	Suicide	Arm (Deltoid)	13 hours	yes	4 days	no	4 days	death
Juluganti.et al 2014[30]	Suicide	Arm	4 days	Yes (12days)	5 days	no	12 days	recovery
Nizami et al 2021[29]	Suicide	Hypochondrial region		yes	21 days	yes	22 days	recovery
Liu. et al. 2013[11]	Suicide	Arm	9 days	no	15 days	no	21 days	recovery
Singh. et al 2015[31]	homicide	Arm	4 hours	Yes (18 days)	_____	yes	28 days	recovery
Zoppellar. et al 1997[39]	Suicide	Leg (quadriceps muscle)	2 hours	Yes (15 days)	18 days	no	32 days	death
Khatua. et al 2023[37]	Suicide	arm	_____	no	7 days	no	12 days	recovery

initially, observation for a more extended period is mandatory. As the decontaminating measures are not applicable in parenteral poisoning, the injection of a relatively small amount may be life-threatening. L-carnitine therapy appears to be a valuable adjunctive treatment in the management of acute ChEI poisoning, enhancing the effectiveness of standard therapies like atropine and oximes, encouraging rapid recovery, and guarding against the development of severe complications such as IMS.

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Ethics Approval and Consent to Participate: The current case report was carried out after approval of the Ethics Committee of the Faculty of Medicine, Alexandria University (IRB number: 00012098, approval serial number: 0201564, FWA number:00018699). Informed consent was obtained from the studied patient. Complete confidentiality was ensured.

Consent for Publication: Not applicable

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