

Atrial Fibrillation with Slow Ventricular Response due to Multi-drug Poisoning – Successful Lipid Emulsion Therapy

■ Nalan KOZACI¹, ■ Songül ÇOLAK¹, ■ Ali Kemal ERENLER¹, ■ İhsan DANIŞ¹, ■ Kenan Ahmet TÜRKDOĞAN¹

¹Department of Emergency Medicine, Alanya Alaaddin Keykubat University, Antalya, Türkiye

Abstract

This case report presents a patient who attempted suicide by ingesting multiple drugs (sertraline, quetiapine, and Tarka [verapamil 240 mg + trandolapril 4 mg]). The patient developed toxicity-related shock, atrial fibrillation with a slow ventricular response, and QTc interval prolongation. Due to persistent hemodynamic instability, intravenous lipid emulsion (ILE) therapy was initiated. Following ILE administration, normal sinus rhythm was restored, the QTc interval returned to normal limits concurrently, blood pressure increased, and clinical stability was achieved. No adverse effects associated with ILE were observed.

ILE may be considered an effective treatment option in the management of life-threatening cardiotoxicity resulting from multidrug overdoses. When administered promptly, lower doses and shorter durations of ILE appear sufficient for successful clinical recovery.

Keywords: Prolonged QT, Lipid Emulsion Therapy, Suicide, Multi-drug Intoxication, Atrial Fibrillation, Multi-drug Poisoning

Introduction

Drug intoxication is one of the most common causes of emergency department admissions. It constitutes 0.8 to 5% of all admissions. Rate of multidrug ingestion for suicidal purposes is quite high. This rate is higher particularly in patients who have died. The reason for high mortality rate in multi-drug ingestion may be linked to interactions between drugs with similar mechanisms of action. A combination of analgesics, sedatives and antidepressants is commonly seen in multidrug ingestions¹⁻³. Intravenous lipid emulsion therapy is a modality used mainly in cardiotoxicity due to lipophilic drug ingestion².

Case Report

A 49-year-old female patient admitted to emergency department with drug ingestion. In anamnesis taken from the patient, it was determined that the patient ingested 28 tablets of sertraline 50 mg, 26 tablets of quetiapine 25 mg, 6 tablets of antihypertensive medicine 240 mg verapamil +

4 mg trandopril 2-3 hours prior to admission. Her general status was moderate with lethargy. Her vital signs were as follows: temperature: 36.5°C, pulse: 62 beats/minute, blood pressure: 100/75 mmHg, SpO₂: % 96, and respiratory rate: 20/minute. Her electrocardiography (ECG) revealed a sinus bradycardia with a heart rate of 57/minute, PR duration of 99 ms and QTc with 452 ms (Figure 1a). A close monitorization was recommended. In follow-up, the patient developed atrial fibrillation and her blood pressure dropped to 70/50 mmHg. On a recent ECG, heart rate was 67 beats/minute, QRS with was 107 ms and QTc was 496 ms (Figure 1b). Following a bolus saline administration, IV calcium gluconate and an inotropic agent (norepinephrine) were initiated. Due to failure of supportive therapy, ILE therapy (1.5ml/kg bolus loading dose) was administered. Following loading dose, a junctional rhythm (HR: 51, QRS: 104 ms, QTc: 457 ms) on ECG (Figure 1c) was determined. Then a continuous ILE infusion (0.25ml/kg/minute) was initiated. In the second hour of infusion, a sinus rhythm (HR: 55 beats/minute, PR:192 ms, QTc: 445 ms, QRS:105 ms) was obtained on ECG (Figure 1d). Her vital signs were as follows: blood

Corresponding Author: Nalan KOZACI **e-mail:** nalankozaci@gmail.com

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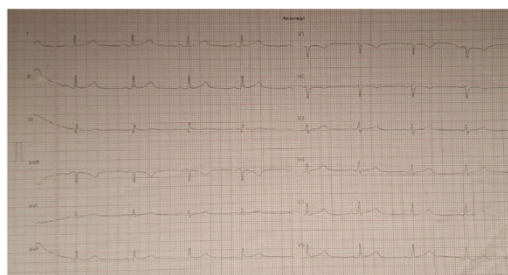


Figure 1a.

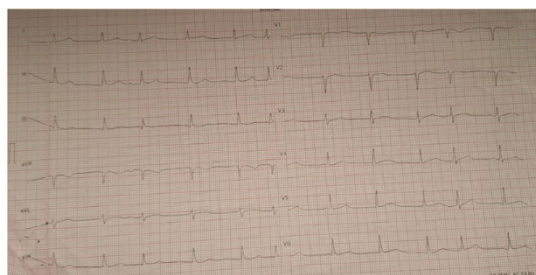


Figure 1b.

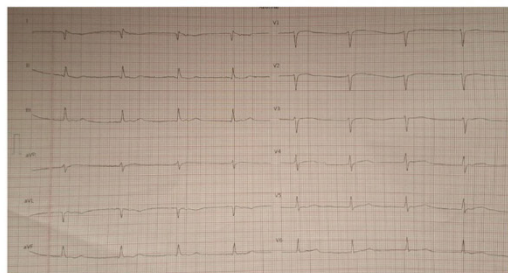


Figure 1c.

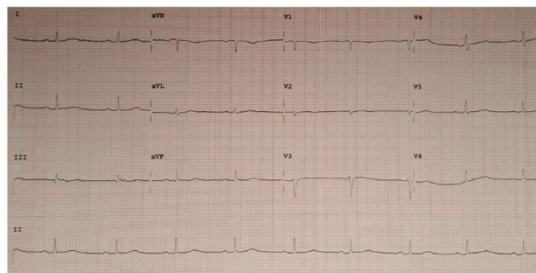


Figure 1d.

Figure 1a: Sinus bradycardia, HR: 57 beats/min, PR: 160 ms, QRS: 99 ms, QTC: 452 ms; **1b:** Atrial fibrillation, heart rate: 67 beats/min, QRS: 107 ms, QTC: 496 ms; **1c:** Junctional rhythm, HR: 51 beats/min, QRS: 104 ms, QTC: 457 ms; **1d:** Sinus bradycardia, HR: 55 beats/min, PR: 192 ms, QTC: 445 QRS: 105 ms

pressure: 100/70 mmHg, pulse: 55 beats/minute. In the third hour of treatment her vital signs improved as blood pressure: 110/70 mmHg, pulse: 73 beats/minute, respiratory rate: 16/minute and SpO₂: %97. A total of 4 hours of ILE therapy was administered. Since the vital signs were stable, ILE therapy was terminated. Any side-effects related to ILE therapy were not determined.

Discussion

Cardiovascular disorders related to drug overdose are associated with high morbidity and mortality. ECG is the first choice of modality for evaluating unwanted conditions. The most useful ECG finding to predict bad outcomes is QTc duration. The risk for cardiovascular involvement is ten-fold higher in case QTc is greater than 500 ms⁴. In a study, Class III antidysrhythmics (sotalol), sodium channel blockers (amitriptyline, diphenhydramine, doxepin, imipramine, nortriptyline), antidepressants (bupropion, citalopram, escitalopram, trazodone), antipsychotics (haloperidol, quetiapine) and the antiemetic serotonin antagonist ondansetron were found to prolong QTc⁴. When SSRIs were compared according to their effects on QTc prolongation, fluoxetine, fluvoxamine and sertraline were found to have similar effects, while paroxetine had the lowest risk in terms of QTc prolongation⁵.

In our case, the patient first developed bradycardia followed by atrial fibrillation with slow ventricular response. Along with arrhythmia, a prolongation of QTc was determined. With changes in ECG, hypotension has emerged. Ketipin overdose is usually known to cause sedation, respiratory failure, hypotension and coma. It also causes prolonged QTc accompanied by tachycardia. Concordantly, QTc prolongation in our case may be related to ketipin overdose. Also, atrial fibrillation developed in our patient

may be associated with katiapin and sertraline. Although arrhythmogenic effect of antipsychotics is well-described, their effect on atrial arrhythmias is not clear. However, in a study, a relationship between high risk for atrial fibrillation development and antipsychotic drugs which are known to have high affinity to cardiac muscarinic receptors⁶. On the other hand, in a meta analysis investigating relationship between antidepressants and atrial fibrillation/ventricular arrhythmias, antidepressant use was found to increase risk of atrial fibrillation⁷.

In Trandolapril intoxication, hypotension and relative bradycardia may occur. The most serious side effects are angioedema and hypotension. These side effects differ trandolapril from other ACE inhibitors. All trandolapril toxicities emerge as a result of ingestion of a trademark called Tarka⁸. Verapamil in Tarka is a calcium channel blocker involved in nondihydropyridine group. Verapamil intoxication causes peripheral vasodilation, hypotension, bradycardia, metabolic acidosis, hyperglycemia, congestive heart failure, lung edema and cardiac arrest^{8,9}. Hypotension and reduction in heart rate in our case may be a result of effects of verapamil and trandolapril.

ILE therapy is recommended when potentially lethal cardiotoxicity develops following lipophilic drug overdose. It is effective particularly in persistent hypotension related with bradycardia¹⁰. One study reported that ILE improved the GCS decreases and QT interval prolongation caused by toxic doses of lipid-soluble neuropsychiatric drugs such as amitriptyline, trazodone, quetiapine, lamotrigine, and citalopram¹¹. However, in a study conducted on poisoning cases in Turkey, it was reported that ILE could be applied as a last resort, especially in patients with poor general condition, regardless of whether they were intoxicated with a lipophilic or hydrophilic substance². This result is especially important for multi-drug poisonings

because in multi-drug poisonings, there may be combinations of hydrophilic and lipophilic drugs.

Although the precise mechanism of action of ILE remains incompletely elucidated, several theories have been proposed. The most widely recognized is the “lipid sink” theory, in which highly lipophilic toxins are sequestered into an expanded plasma lipid phase, thereby rapidly reducing their free (unbound) plasma concentration and limiting tissue distribution. Secondly, ILE improves blood pressure and cardiac output through direct cardiokinetic effects. Additionally, lipids are thought to exert cardioprotective effects through multiple biochemical pathways, including enhancement of myocardial energy substrate utilization, restoration of mitochondrial function, improvement of calcium homeostasis, and reduction of oxidative stress. Despite these insights, the exact contribution and dominance of each pathway for specific toxins remain uncertain, and the definitive biochemical mechanisms are still not fully understood^{12,13}.

The recommended ILE protocol consists of an initial bolus of 1.5 mL/kg of 20% lipid emulsion administered over 2–3 minutes, followed by a maintenance infusion of 0.25 mL/kg/min. If cardiovascular instability persists, repeat bolus(es) of 1.5 mL/kg and doubling of the infusion rate (up to 0.5 mL/kg/min) may be considered¹³.

Our case was intoxicated due to multi-drug consumption. As a result of the combined effects of the drugs she ingested; hypotension, AF, bradyarrhythmia and prolongation of QTc interval developed. Saline and IV calcium are used to treat cardiac side effects in verapamil toxicity. Hyperinsulinemic euglycemia treatment and glucagon administration are recommended to improve cardiac inotropy and glucose utilization. Glucagon has inotropic effects but is not effective in improving bradycardia^{8,9}.

In our case, bradyarrhythmia and hypotension did not improve despite IV saline bolus, calcium gluconate and high dose inotropic agent treatment. ILE is successfully used in the treatment of neuropsychiatric drugs and calcium channel blocker toxicity^{8,10}. ILE therapy was initiated in our patient, whose clinical condition remained unstable. Following the ILE bolus, a junctional rhythm was observed on the follow-up electrocardiogram (ECG). At the second hour of the maintenance infusion, sinus rhythm was restored; however, bradycardia persisted. By the third hour of ILE infusion, heart rate normalized. Concomitantly with the re-establishment of normal sinus rhythm, the QTc interval returned to within normal limits. A simultaneous increase in blood pressure was noted, and the patient's clinical status stabilized. Lipid treatment was applied for a short time, 4 hours, and there was no need to repeat it. Our patient was discharged with a full recovery without any sequelae.

Conclusion

In acute drug overdose and multi-drug intoxication; hypotension, shock, cardiac rhythm and conduction disorders that can be fatal may develop in cardiovascular deterioration. When there is no response to other treatments,

ILE treatment may be an option in the resuscitation of these patients. When ILE is given immediately, shorter periods of administration and lower doses are sufficient for treatment.

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