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Case Report

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Successful treatment with intravenous lipid emulsion of severe tricyclic antidepressant intoxication

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ABSTRACT

An overdose of amitriptyline has neurological and cardiovascular side effects (psychosis, lethargy, coma, hypotension, and dysrhythmia). A 19-year-old female patient was admitted to our emergency service after a suicide attempt by amitriptyline ingestion of unknown amount. Her follow-up demonstrated deteriorating consciousness and vital signs, she had a generalized tonic-clonic seizure, and her electrocardiogram (ECG) revealed QRS interval widening followed by a ventricular tachycardia (VT) attack. The patient was treated with intravenous (IV) diazepam (Diazem®) to control the epileptic seizure, IV lidocaine (Aritmal®) to stop the VT attack, and IV sodium bicarbonate due to QRS interval widening. The patient was given norepinephrine bitartrate (Steradine®) and dopamine hydrochloride (Dopamine®) due to low blood pressure. Intravenous lipid emulsion (ILE) therapy was started due to the uncontrolled rhythm and hypotension. Here, we present a case report of a comatose patient with dysrhythmia due to amitriptyline overdose who responds to ILE therapy.

1. Introduction

Amitriptyline is the most commonly used tricyclic antidepressant (TCA) for suicide (Caksen et al., 2006). TCA overdose results in a toxicity syndrome characterized by neurological and cardiac side effects. Poisoning-related clinical manifestations vary from mild muscarinic effects and severe cardiotoxicity due to sodium channel blockade to death. Change in mental status is the most common symptom after TCA toxicity (Bateman et al., 2005). The general treatment approach to TCA toxicity includes gastrointestinal decontamination, fluid replacement, sodium bicarbonate, and magnesium sulphate in case of resistant dysrhythmias. Although there is today no consensus on the use of lipid emulsion therapy for the treatment of TCA toxicity, the current literature includes case reports on its usefulness (Agarwala et al., 2014; Levine et al., 2014; Ozcan et al., 2014; Odigwe et

al., 2016). Here, we present a case report of a comatose patient with dysrhythmia due to amitriptyline overdose who responds to ILE therapy.

2. Case report

A 19-year-old female patient was admitted to our emergency department due to deterioration of her general condition after a suicide attempt by amitriptyline ingestion of unknown amount. She underwent gastric lavage followed by activated charcoal, her follow-up demonstrated deteriorating consciousness and vital signs, she had a generalized tonic-clonic seizure, and her electrocardiogram (ECG) revealed QRS interval widening followed by a ventricular tachycardia (VT) attack. The patient was treated with intravenous (IV) diazepam (Diazem®) to control the epileptic seizure, IV lidocaine (Aritmal®) to stop the VT attack, and IV sodium bicarbonate (1-2 mEq/kg) due to QRS interval widening. The patient was given norepinephrine bitartrate (Steradine®) at 30 mcg/minute and dopamine hydrochloride (Dopamine®) IV at 30 mcg/minute of serum physiologic solution due to low blood pressure. The patient was intubated because of her GCS score was 3/15. Despite the positive inotropic support, her vitals were blood pressure 70/40 mmHg, and pulse 180 beats/min. When the ECG of the monitored patient revealed VT (Fig. 1), three rounds of synchronized cardioversion of 100-100-200 joules were performed on the hypotensive patient with ongoing VT despite IV lidocaine administration (Fig. 2). The laboratory tests were normal. The patient was given IV magnesium sulphate 2 g due to resistant VT. ILE therapy was started due to the uncontrolled rhythm and hypotension. She was given a bolus of 100 mL (1.5 mL/kg) IV 20% lipid emulsion followed by an infusion of 400 mL. The patient was extubated 48 hours after her admission to the hospital and there was no need for vasopressors on the third day of admission. The patient who had stable vital signs and no additional problem was discharged with full recovery on the fifth day of admission in line with psychiatric recommendations (Fig. 3).

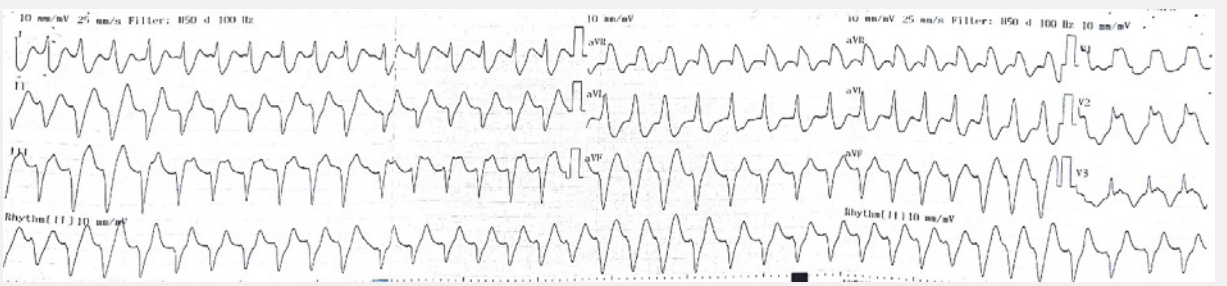

Fig. 1. Wide QRS tachycardia (Ventricular tachycardia) on admission.

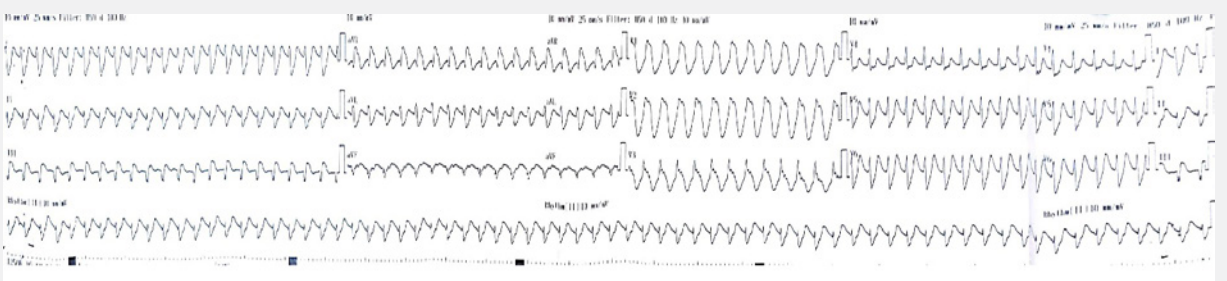

Fig. 2. Electrical cardioversion applied for ventricular tachycardia.

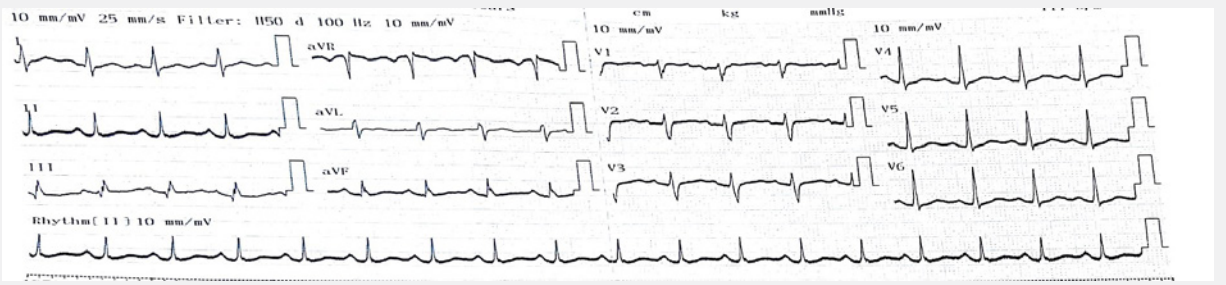

Fig. 3. Normal electrocardiogram after treatment.

3. Discussion

An intentional overdose of tricyclic antidepressants (TCAs) is a major cause of death. TCAs produce their pharmacological effects by inhibiting presynaptic serotonin and norepinephrine uptake. TCAs are rapidly absorbed from the intestine and reach high plasma levels within 2-8 hours (Odigwe et al., 2016). Our patient had altered mental status (coma), epileptic seizure, hypotension and resistant dysrhythmia approximately 3 hours after the overdose of amitriptyline.

Cardiotoxicity is the main cause of death in TCA overdose, mainly due to myocardial sodium channel blockade (Levine et al., 2014). Lipid emulsion therapy is a potentially new treatment used to reverse the cardiotoxicity caused by TCA overdoses (Chan et al., 2010; Levine et al., 2014). Sodium channel blockade caused by TCAs prolongs the refractory period and delays atrioventricular (AV) node conduction. This delay causes QRS widening, and QTc and PR prolongation on the ECG (Thanacoody et al., 2005; Aksakal et al., 2010). A QRS duration longer than 100 msn is an indication for bicarbonate therapy in cases of TCA overdose (Odigwe et al., 2016). QRS and QT intervals of our patient with resistant VT were 200 and 360 msn on the admission ECG. PR, QRS, QTc and QT/QTc intervals on the discharge ECG were within the normal range and 166, 94, 392 and 328/392 msn, respectively.

Today, no specific method is available in clinical practice to determine that sodium bicarbonate proves unsuccessful and other treatments should be initiated, thereby presenting challenges for clinicians to decide. Clinicians tend to initiate other treatments depending on disease severity and the response of electrocardiographic and hemodynamic parameters to sodium bicarbonate (Odigwe et al., 2016). We started ILE treatment for our patient who did not respond to other therapies including electrical cardioversion and had hemodynamic impairment and resistant VT attack. The mechanism of action of lipid therapy is not fully understood (Agarwala et al., 2014; Odigwe et al., 2016). Its role in humans remains uncertain (Odigwe et al., 2016). ILE therapy reduces bioavailability by forming a "lipid sink", i.e. trapping lipophilic agents in an expanded plasma lipid compartment, thereby inhibiting the effect of TCA and prevents toxicity (Chan et al., 2010). According to an alternative theory, ILE improves myocardial free fatty-acid availability by reversing the shift from lipid to glucose metabolism in stunned myocardium. ILE may also prevent the inhibition to oxidative phosphorylation in toxic myocardium (Levine et al., 2014). In patients with cardiotoxicity resistant to other treatment modalities, it is recommended to administer a 100 mL IV bolus injection of 20% lipid emulsion (1.5 mL/kg) for 2-3 minutes and a continuous IV infusion of 0.25 mL/kg/min (18 mL/min) at a total dose of 10 mL/kg (American College of Medical Toxicology, 2011). We initiated ILE therapy for our patient in the ICU due to resistant VT. Our patient received a total of 1000 mL IV lipid emulsion throughout her entire hospital stay.

The current literature includes a considerable number of case reports on the success of ILE in the treatment of TCA-induced cardiotoxicity. ILE therapy may be beneficial in the management of severe amitriptyline toxicity characterized by resistant hemodynamic instability and malignant dysrhythmias in case of failure to respond to other treatment modalities. Early-onset relative high-dose ILE therapy may be life-saving in patients with resistant hemodynamic instability due to TCA-induced severe cardiotoxicity.

Conflict of Interests

The authors declare that there is no conflict of interest.

Financial disclosure

The authors declare that no funding was granted for this research.

Statement of informed consent

A written informed consent form obtained from the patient at the time of discharge.

Author's contributions

HUA, YC, NUA conceived the study and design the trial, supervised the conduct of the trial, data collection and drafted the manuscript. HUA, YC, MU under took recruitment of participating patient. HUA, NUA managed the data, including quality control. HUA, NUA, YC supervised the conduct of the trial and data collection. HUA, MU, YC, NUA drafted the manuscript and managed the data, including quality control and all authors contributed substantially to its revision.

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