ST Elevation in Tricyclic Antidepressants Toxicity: A Case Report

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Abstract

Of all antidepressants, tricyclic antidepressants (TCAs) are the most toxic drugs and they are often used for suicide attempts. An 18-year-old female was admitted to the emergency department after having taken 30 tablets of Nortriptyline 50 mg in a suicide attempt**.** A twelve-lead electrocardiogram revealed right bundle branch block in leads V_1, V_2, and V_3 as well as deep S in leads İ, V_5, and V_6, and tall R in AVR, which meant extreme right axis and ST elevation in leads V_2 and V_3: that raised the suspicion of the Brugada syndrome. ST elevation after TCA toxicity is a rare medical condition, and our literature review failed to find any relevant reports. ***(Iranian Heart Journal 2012; 13(3):43-45) .***

Keywords: TCA ■RBBB■ Brugada

Introduction

Tricyclic antidepressants (TCAs) were one of the most important causes of mortality resulting from poisoning until 1993. Since then, TCAs have continued to be responsible for more deaths per prescription than all the other antidepressants put together [(1)]. In 2006, about 6000 cases of cyclic antidepressant overdose were reported, with 4% resulting in serious adverse outcomes including death [(2)]. Cyclic antidepressants have a narrow therapeutic window, which increases their likelihood for toxicity [(3)].

In the differential diagnosis of comatose patients with a history of depression and/or attempts, intoxication should be taken into consideration. TCAs alone or in combination with other drugs are often used in intentional drug overdosing. Serious neurological (coma and convulsions), anticholinergic, and cardiovascular (hypotension and ventricular arrhythmias) effects may occur; therefore, patients are frequently admitted to the Intensive Care Unit [(4)].

An electrocardiogram (ECG) is an essential diagnostic tool in assessing the clinical severity of overdose because impaired conduction can progress into arrhythmias with cardiovascular collapse. However, ECG can never be utilized as the sole method of risk determination [(5)]. We herein report ECG abnormalities, especially ST elevation, due to TCA poisoning in the clinical setting.

Case Presentation

An 18-year-old female was admitted to the emergency department after having taken 30 tablets of Nortriptyline 50 mg in a suicide attempt, half an hour before admission. On arrival at the emergency department, the patient was lethargic with involuntary movements, the EMV score was $E_{\Pi}M_{\Pi}V_{\Pi}$, and the pupil reactions were normal. She demonstrated no lateralization and her reflexes were symmetric. No meningeal signs were noted. On physical examination, the patient had a blood pressure of 80/60 mm Hg, pulse rate of

Received November 2012; Accepted for publication December 2012

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100 beats per minute, respiratory rate of 24 beats per minute, and temperature of 37.1°C. O2+Naloxane+Dextrose was administered for her, but her condition did not differ. She was thereafter endotracheally intubated and mechanical ventilation was commenced.

A twelve-lead ECG revealed right bundle branch block in leads V_1, V_2, and V_3 as well as deep S in leads İ, V_5, and V_6, tall R in AVR, which denoted extreme right axis and ST elevation in leads V_2 and V_3: the Brugada syndrome is the first thing to spring to mind. ST elevation after TCA toxicity is a rare medical condition, and we did not find any related reported cases in the literature(Figures 1 and 2).

The technique of plasma TCA level measurement was not available in our hospital. Bicarbonate infusion (1 meq/kg) was performed for the patient, who was subsequently transferred to the Intensive Care Unit for observation. Twenty-four hours later, the patient was extubated and forty-eight hours afterward, she was transferred to the internal medical ward.

Figure 1.

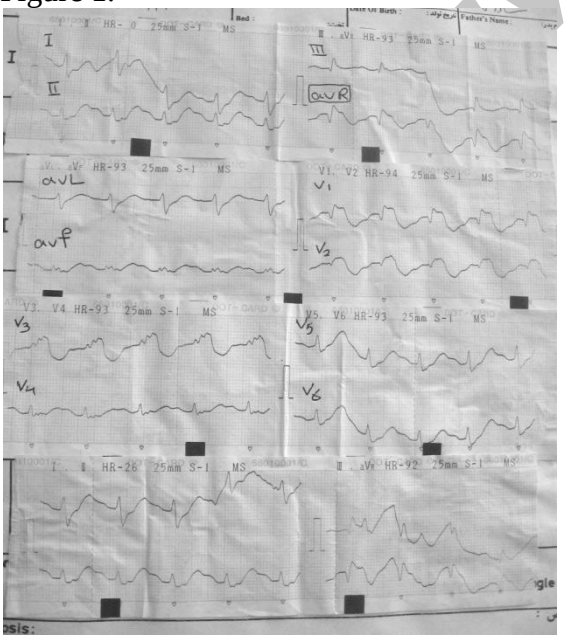
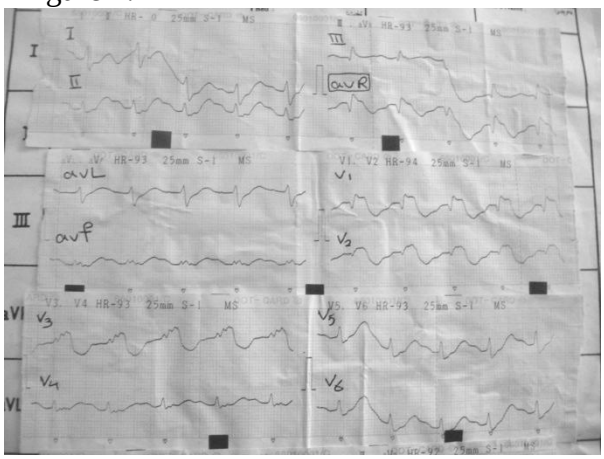


Figure 2.



Discussion

Of all antidepressants, TCAs are the most toxic drugs and they are often used for suicide attempts. Diagnosis of TCA intoxication can be very difficult in that the clinical signs, however severe, are non-specific. The mechanism of TCA toxicity is probably pharmacological actions: anticholinergic; vascular α1-antiadrenergic action; adrenergic reuptake inhibition at terminals; fast sodium channel blockade; and quinidine-like effect in the heart. Serious anticholinergic effects include decreased bowel movements, hyperthermia causing rhabdomyolysis, urinary retention, renal failure, and confusion. Cardiovascular and neurological effects are also very common. By inhibiting sodium channels, TCAs can delay the propagation of depolarization and repolarization in the myocardium, leading to the prolongation of PR, QRS, and QT intervals. Furthermore, AV blocks, bundle branch blocks, severe ventricular tachycardia, and sinus tachycardia may occur [6]. Our patient presented with right bundle branch block and ST elevation.

Hypotension may be the result of a combination of diminished myocardial contractility (inhibition of fast sodium channels), vascular dilation (blockade of α_1-adrenergic neurotransmitters), and norepinephrine (NA) reuptake inhibition (can lead to NA depletion) [7,8].

An ECG is an essential diagnostic tool in assessing the clinical severity of overdose

because impaired conduction can progress into arrhythmias with cardiovascular collapse. However, it can never be used as a sole method of risk determination. The accuracy of the ECG is influenced by the time of drug ingestion. Directly after intake, the ECG is usually still normal, with abnormalities developing after several hours. Repeat ECGs are necessary when TCA intoxication is suspected.

Acknowledgment
We are sincerely indebted to all the hospital staff who helped us to write this manuscript.

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