

Intralipid Therapy Use in Tricyclic Antidepressant Overdose A Case Report

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Introduction

The use of lipid emulsion therapy (LET) has been well established in local anesthetic systemic toxicity (LAST). The evidence for the use of this therapy in tricyclic antidepressants (TCA), however, is scant. We describe a case where use of LET in a patient cardiac arrest from massive TCA overdose resulted in return of spontaneous circulation (ROSC).

Case Presentation

A 19 year-old girl presented to the Emergency Department (ED) via ambulance with pulseless ventricular tachycardia (VT). She was previously prescribed amitriptyline for depression. The patient was found by her mother unconscious with a partially empty pill bottle of amitriptyline about an hour before. A pill count revealed a total dose of amitriptyline 1.25g. She was initially drowsy but developed pulseless VT in the ambulance 10 minutes prior to arrival at the ED.

Despite pre-hospital defibrillation and cardiopulmonary resuscitation (CPR), the patient was still in pulseless VT in ED. In addition to conventional resuscitative measures (ie. continued CPR, defibrillation, IV adrenaline, and intubation), IV sodium bicarbonate boluses were administered.

Despite repeated boluses of sodium bicarbonate totalling up to 500mls, the patient still remained in pulseless VT. LET was administered at a dose of 1.5ml/kg bolus, following which the patient had ROSC. Although she was still intermittently in VT, her blood pressure ranged from 99-148/74-95 unsupported in the ED.

During her stay she became gradually more hemodynamically unstable and later developed deep vein thrombosis with compartment syndrome of the leg requiring a fasciotomy. This was then complicated by reperfusion syndrome and recurrent cardiac arrests with worsening acidosis and hyperkalemia. She eventually died on day 2 of ICU admission.

Discussion

To our knowledge, the use of LET for TCA overdose is limited to case reports and animal studies. While the exact mechanism is of some debate, a popular theory is that of the 'lipid sink', where lipophilic drugs are drawn out of the target tissue and toxicity is reversed. As amitriptyline is a highly lipid-soluble molecule, it is purported that LET can work in a similar manner to reduce its toxic effects.

Although our patient eventually died, there were other complicating aspects which may have contributed to this. In fact, she achieved ROSC and re-gained good cardiac output shortly following intralipid therapy. Hence, it is likely that intralipid therapy is effective at reversing to a large extent the cardiotoxic effects of TCAs.

In a systematic review, potential adverse effects of LET were found to include acute kidney and lung injury, venous thromboembolism, hypersensitivity, fat embolism, fat overload syndrome, pancreatitis, extracorporeal circulation machine circuit obstruction, and increased susceptibility to infection. Despite this, LET is a fairly last-ditch measure in which the benefit to the patient would likely outweigh possible risks.

Our case concurs with other case reports that suggest LET may have a role as an adjunct in severe TCA overdose. However, more evidence is needed before its routine use can be recommended.

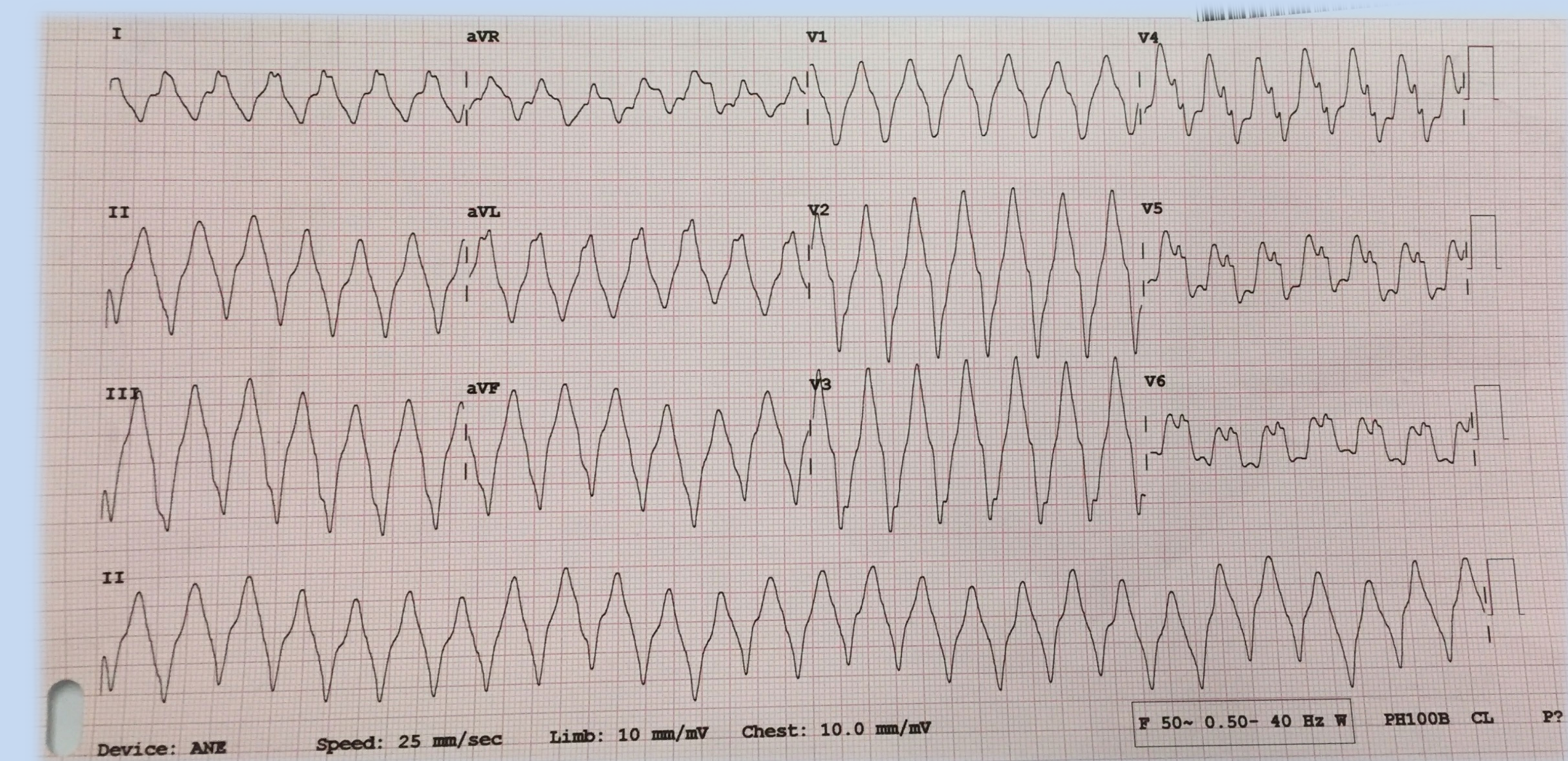


Fig 1 : Patient's ECG post return of spontaneous circulation