Another 'deliberate self-harm': attempting suicide by overdose

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Emergencies can spring up at any time and in many incarnations. Are you adequately equipped to deal with them? Each month we present a case study in emergency medicine based on real cases and events. Would you have been able to help this patient?

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As an experienced GP routinely doing shifts in a busy city emergency department, as well as from personal experiences, you are very aware of the issue of DSH – deliberate self harm. It is mystifying to you that suicide is responsible for 22% of deaths among young people in such a great place to live as Australia. Boys kill themselves some four times as often as girls but girls make up to ten times more suicide attempts than boys.

Today, a drowsy, well-dressed 18-year-old girl is brought in by ambulance – you and the team methodically and in a semi-detached manner begin to assess yet another overdose.

You go through the 'ABC' of resuscitation (airway, breathing, circulation). At the moment the patient is protecting her airway but as the ambulance staff hand you several empty blister tablet packets of a tricyclic antidepressant as well as an empty bottle of diazepam tablets you realise this girl is in big trouble.

The nurses and doctors automatically do the routine observations and set the patient up for a detailed assessment. The initial approach is summarised by the mnemonics of 'ABC' and 'DON'T':

- Dextrose
- Oxygen
- Naloxone
- Thiamine.

After a quick check in the reference book you become even more worried about this patient.

On its own, dothiepin has anticholinergic properties as well as central sedative properties. Yes, it is well-absorbed, with some 80% protein-bound. It is excreted mainly in the urine. Dothiepin's half-life is biphasic with a mean of some 50 hours. As with most tricyclic antidepressants, toxicity is related to its anticholinergic effects on the:

- gastrointestinal tract – gastric stasis, ileus
- central nervous system – mydriasis, agitation, delirium, hyperpyrexia, convulsions, coma, respiratory depression
- cardiovascular system – tachycardia and arrhythmias (often the cause of death).

The reference manual clearly states that death has occurred with 0.75 g (30 tablets). You think this patient has taken at least 40 to 50 tablets at once (that is, 1.0 to 1.25 g) and you can't be sure how many tablets were taken routinely before the overdose.

The team inserts intravenous lines. The initial ECG shows a tachycardia of 120 beats per minute with definite widening of the QRS complex. Both findings are clear signs of cardiac toxicity with the accompanying threat of ventricular fibrillation.

The patient's conscious state decreases and soon there is no longer a gag reflex protecting her airway. After good preoxygenation, she is sedated, paralysed, and intubated. Blood is

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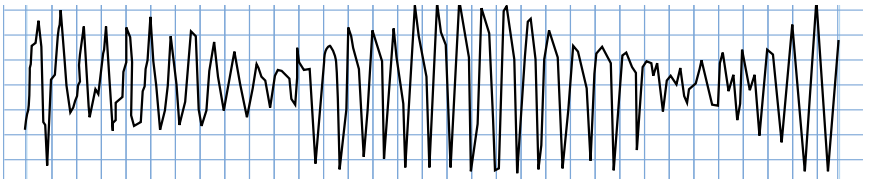

Figure. Torsade de pointes. Resembling both ventricular tachycardia (VT) and ventricular fibrillation (VF) in appearance, torsades de pointes has been described as polymorphous VT, with irregular wide bizarre QRS complexes with alternating positive and negative polarity. Recognition is important, as treatment is different from the usual monomorphic form of VT.

taken for routine testing, including paracetamol levels. A nasogastric tube is inserted to try aspirate as much of the gastric contents as possible (with a sample kept for the lab, if needed). Then, charcoal and mannitol are infused nasogastrically and metaclopramide is given intravenously to promote elimination of the tricyclic antidepressant.

As the mainstay of all overdose management is meticulous physiological support, the patient is assessed and accepted to intensive care.

It turns out that the patient got the prescription for dothiepin when she saw her psychiatrist yesterday. A major overdose is confirmed.

In overdoses such as this one, anything that can worsen the arrhythmogenicity is greatly feared. Two obvious concerns are:

- hypoxia
- acidosis.

Initial arterial blood gases measured shortly after intubation show an acid pH of 7.2. There is some discussion about the use of physostigmine, 2 mg intravenously, in order to attempt to reverse the anticholinergic effects of dothiepin. However, this is, as usual, a judgment call, as even with the administration of bicarbonate there are, at times, new problems created, and polypharmacy can compound the problems. It is decided to see if good ventilation, oxygenation and hydration will, in themselves, reverse the acidosis – they do.

After some hours, the tachycardia and QRS changes begin to resolve, thus relieving the fear of ventricular fibrillation, often with the classic '*torsades de pointes*' – that is, where the electrical axis of the VF swings around giving a harmonic but deadly pattern on the ECG tracing (Figure).

Upon your enquiry the next week you find the patient did well and was discharged home. You collar a passing psychiatry registrar and ask: 'Why are tricyclics still used in practice when there are now several very effective newer antidepressants that are very much safer in an overdose, – a situation that is always a risk in depressed patients?'

The answer back is that while tricyclic antidepressants are no longer used first line in depression, they may still be specifically indicated on occasion.

You reply that from your point of view, the less tricyclic antidepressants are used the better. In a world where the incidence of depression seems to be increasing, you don't need the additional stress of worrying about the very real chance of death in depressed patients who take an overdose of a tricyclic antidepressant.

The registrar agrees with you and also points out that when a patient takes a drug overdose, the risk of recurrence is high. Resuscitation after an overdose is vital but only half the job; further appropriate psychiatric intervention and monitored follow up (i.e. to ensure the patient keeps appointments) is needed to reduce the risk of recurrence. MT