ED QUICK QUIZ

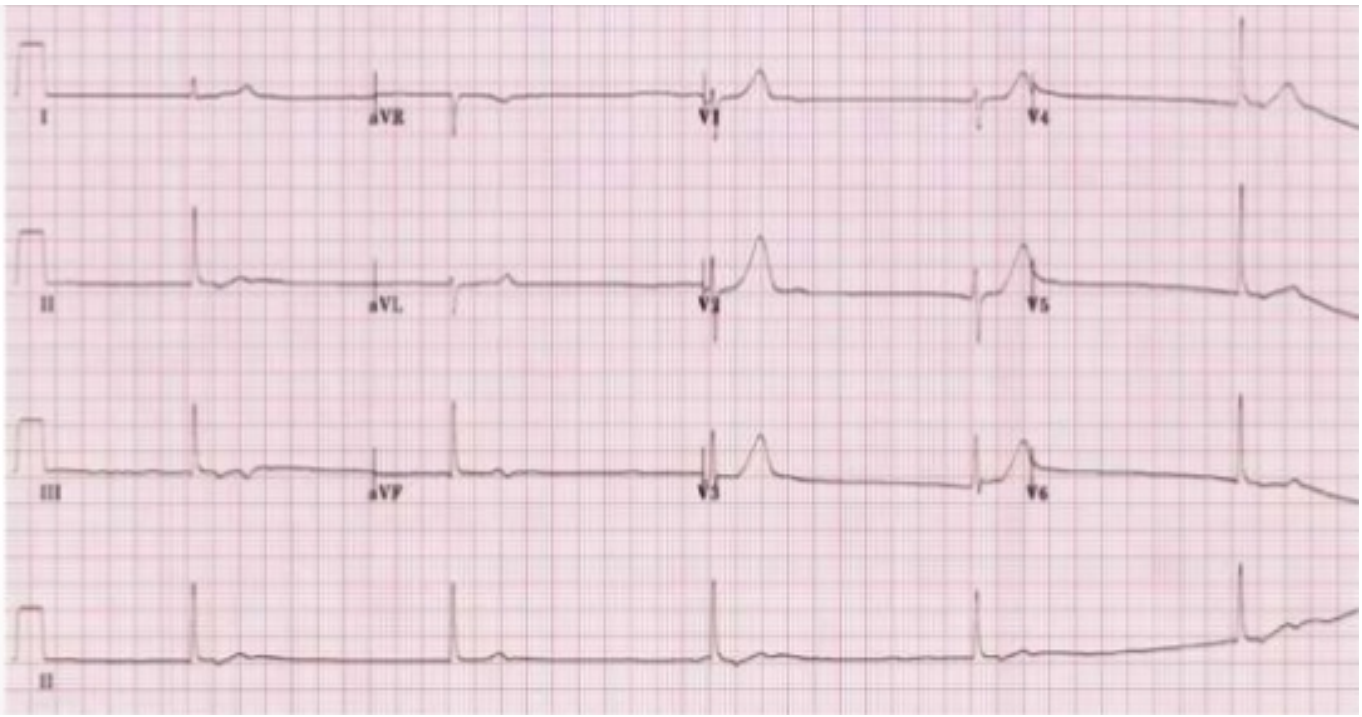
WHAT IS THE DIAGNOSIS?

BACKGROUND

A 52 yr old man is admitted after an overdose of verapamil tablets. He is currently taking verapamil 240mg SR daily and took 20 tablets 2 hours ago to try and end his life. He weighs 75kg. PMH- CABG 2007, asthma well controlled.

O/e
He is alert with oxygen sats of 95% in air but he is clammy and pale.
His heart rate is 35 and BP is 78/45.

An ECG is performed and shown below.



QUESTIONS

1. What is the mechanism of action of verapamil?
2. How would you manage this gentleman?
3. What are the main causes of death from CCB overdose?

ANSWERS & DISCUSSION

1. Verapamil is a calcium channel blocker (CCB). CCBs block L-type voltage sensitive calcium channels in the cell membrane. They work on the myocardial cells, the cells within the conducting system of the heart and also the vascular smooth muscle. They reduce myocardial contractility, supress electrical impulse propagation through the SA and AV node and reduce systemic vascular resistance. The dihydropyridine CCBs like amlodipine and nifedipine have more affinity for inactivated calcium channels which are found in vascular smooth muscle rather than the heart.

2. Your A-E examination finds bradycardia with hypotension. The patient is on a cardiac monitor. You give 500mcg of atropine which produces some benefit with a HR of 65 and BP 100/50. You have taken blood for a VBG and sent formal bloods including U&Es, calcium, glucose, magnesium, paracetamol and salicylate levels.

   This man has taken in total 4800mg verapamil, which is well over the amount reported to cause toxicity on toxbase. Given ingestion was 2 hours ago and the tablets are slow release you consider late administration of activated charcoal or bowel irrigation. You discuss these with the Consultant and patient and opt for activated charcoal.
   You follow toxbase advice and give 10% calcium chloride 0.2ml/kg up to 10ml over 5 minutes and consider an insulin dextrose infusion as this has been shown to improve cardiac contractility particularly in acidotic patients.
   Verapamil toxicity poses a risk of torsade de pointes. The QT interval (start of QRS to end of T wave) on the above ECG is 12 small squares. 12x 40msec= 480msec. Checked against the normogram on toxbase– this is just on the high risk borderline so you give 2g IV Magnesium over 10-15 minutes. When bloods are back you will check potassium, magnesium and calcium are at the upper end of normal and correct if not to try and prevent torsade de pointes.
   Your patient is admitted to CCU for ongoing management. Slow release verapamil reaches plasma peak at 4-8hrs with a half-life of approximately 5-8 hours, but this is often prolonged in overdose.
   If bradycardia returns, more atropine can be given, but discussion with cardiology with regards to ongoing management as sometimes dobutamine and isoprenaline are used or external or temporary pacing. You would also discuss with local poisons info service on toxbase.

3. Features of verapamil and diltiazem overdose are profound cardiac depression causing hypotension and SA and AV block causing bradycardia, torsade de pointes and convulsions.

References

ECG from Andy Steval Toxicology and the ECG 16/3/16
Toxbase accessed 9/5/18
BNF 74