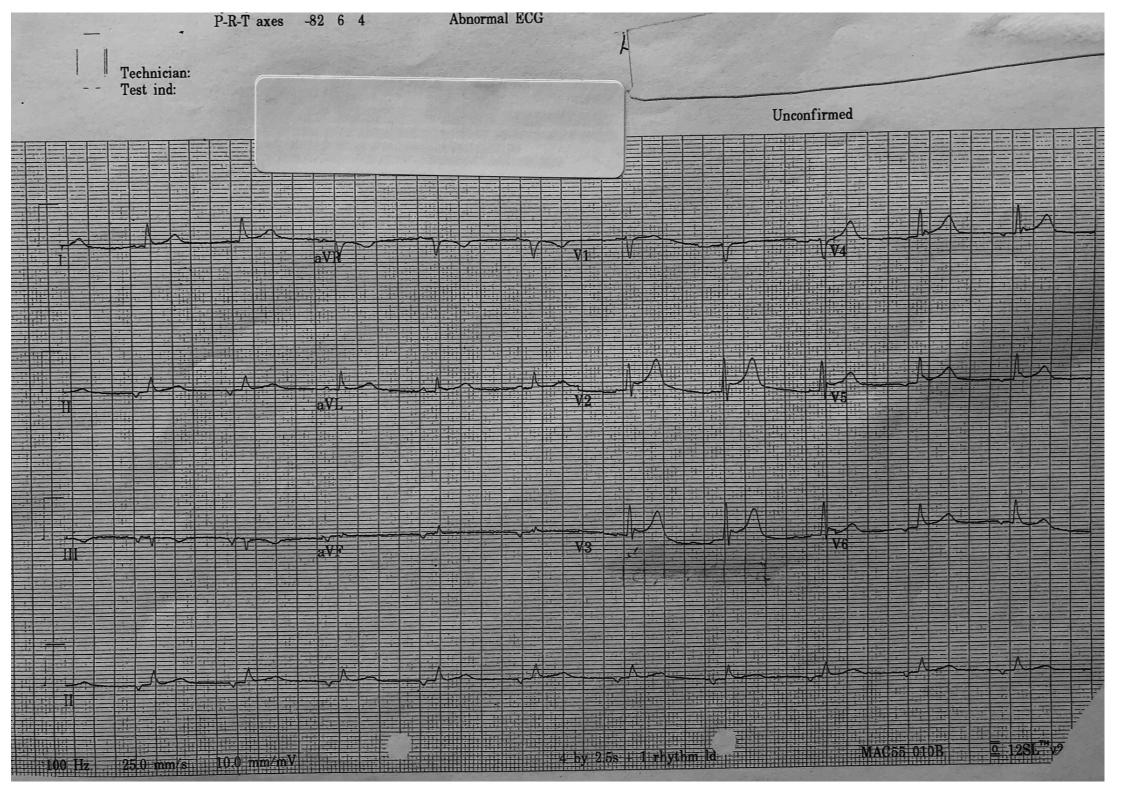
QUIZ 13th Nov 2019 (answers below)

1.	What are the clinical features of quetiapine toxicity?
2.	How would you manage the hypotension of quetiapine toxicity?
3.	What are the ECG changes in tricyclic antidepressant toxicity?
4.	What is the role of sodium bicarbonate in tricyclic antidepressant toxicity?
5.	Describe and interpret the following ECG.



QUIZ answers 13th Nov 2019

1. What are the clinical features of quetiapine toxicity?

<3g Mild-Mod sedation
Sinus tachycardia, which can exceed 120/min
Minor QT prolongation – no reports of Torsades

>3g Increasing sedation leading to coma Hypotension (antagonism at peripheral α_1 receptors) Delirium (due to central anticholinergic action) Seizures

2. How would you manage the hypotension of quetiapine toxicity?

Fluid bolus 20mL/kg crystalloid. If response to fluid resuscitation is inadequate, use Noradrenaline. Of note, Adrenaline may paradoxically exacerbate the hypotension. This is due to excessive β_2 vasodilation.

3. What are the ECG changes in tricyclic antidepressant toxicity?

Fast sodium channel blockade:

- QRS widening
 >100msec is associated with seizures
 >160msec is associated with ventricular dysrhythmias
- Right axis deviation of the terminal QRS
 Terminal R wave >3mm in aVR
 R/S ratio > 0.7 in aVR

Potassium channel blockade:

QT prolongation

Anticholinergic effect:

Tachycardia

4. What is the role of sodium bicarbonate in tricyclic antidepressant toxicity?

Sodium bicarbonate provides both a sodium and a bicarbonate load.

Bicarbonate

- Raises serum pH which improves fast sodium channel function
 This effect is maximal at pH 7.5- 7.55
- Elevated pH reduces the proportion of drug in un-ionised form
 This reduces its ability to cross cell membranes and hence reduces the proportion of drug that distributes to tissue compartments.

Sodium load

Separate and additive positive effect on sodium channel function

Sodium bicarbonate is recommended in ventricular dysrhythmias, hypotension and cardiac arrest as a result of tricyclic antidepressant toxicity. Doses of 2mmol/kg IV are repeated until haemodynamic stability is achieved. In the intubated patient, alkalinisation can be achieved with hyperventilation. The pH aimed for is 7.5 – 7.55.

5. Describe and interpret the following ECG.

Rate Regular 64/min

P waves All present and conducted

Inverted inferiorly (II, III and aVF) and laterally (V4-6)

PR interval Normal

QRS Narrow

Normal axis (~0 degrees) No pathological q waves

Counterclockwise rotation (aka early transition in chest leads)

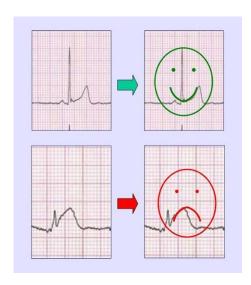
- but no evidence of RV hypertrophy)

ST segments Early repolarisation evident in V2-4 (see diagram below)

T waves NAD

QTc Not prolonged

→ Coronary sinus rhythm Early repolarisation



Early repolarisation (happy) Vs. Pathological ST elevation (sad)

Chest lead rotation

