GUIDE TO INTERPRETING ECG 1.

This ECG highlights some very important findings that indicate a high - risk situation and a time-critical condition.

If you would like to go back the ECG again before reading the full answer – look at

- Rate and rhythm
- QRS morphology
- ST segment deviations and T wave morphology
- Various intervals including the PR interval as well as the QT interval.

This ECG shows a normal sinus rhythm with a rate of around 90-100 bpm and also shows classic features of HYPOKALAEMIA: Why? Read on!

- **T wave inversions**: Almost diffusely present but most prominent in lateral pre-cordial and inferior leads. The T waves look as if someone is actually pushing them downwards!

  If you track T waves with decreasing levels of serum potassium, you will notice flattening of T waves followed by bi-phasic T waves (down – up morphology), ST - depressions and then followed by deeply inverted T waves. At some stage during these progressive changes, U waves will also start appearing after the T waves and as you might already know, U waves are almost pathognomonic of hypokalaemia in the right clinical settings – read on!
  What are the other important causes of T – wave inversions? Time to do some research!

- **U waves**: If you haven't already noted – leads V2 and V3 display U waves after the T waves.

- **Pseudo-prolonged QT interval**: This is called a "pseudo"prolonged QT interval in this case because in actual fact, this is a fused QT and U wave giving the appearance of a prolonged QT interval. The QTc in this ECG is 570 msecs! (see below)

  *We should actually call this a QU interval but as I said before, U waves may not always be seen clearly in hypokalaemia so its best to refer to this as QT prolongation rather than QU interval. The QT interval varies with the heart rate and that is why it is recommended to "correct" the QT interval to the heart rate – the corrected QT interval or **QTc** as it is called. There are various formulae to calculate the QTc which are complex and time consuming but thanks to pre-prepared nomograms, we can now look up the QTc for a given QT interval and heart rate almost instantly. It is good to have a little QTc nomogram lanyard on you if you routinely read ECGs in your clinical practice. The machine - read QT intervals may underestimate the actual QT prolongation.*

As a rule of thumb, between heart rates of 60-100, a QT interval that is more than half the R-R interval is considered prolonged.

QTc > 450 msec is considered prolonged although acceptable ranges are different for males (440-445 msecs) and females (435 – 440 msecs). QTc > 550 msecs puts a patient at significant risk of malignant arrhytmias.

Can you think of any other causes of a prolonged QTc, besides hypokalaemia?

Another common finding with hypokalaemia, although not seen in this case, is **increased P wave amplitude.**

If I now tell you that hyperkalaemia produces exactly the **opposite** sequence of ECG changes seen with hypokalaemia, can you try and list those changes?

Coming back to this patient:

We know that prolonged QTc puts patients at higher risk of dangerous arrhythmias – most notable Torsades de pointes and VF. Frequent ventricular ectopic beats and an R – on - T phenomenon then often triggers a dangerous arrhythmia. This patient subsequently had a cardiac arrest due to Torsades de pointes.

Whenever looking at a patient's potassium levels, always interpret them along with the patient's pH. And bear iin mind, patient's K levels might fluctuate minute to minute especially if the pH is abnormal. If the pH decreases (i.e patient becomes acidotic), the K levels will increase by about 0.6 mmols per 0.1 increase in pH. The exact opposite happens if pH increases, that is, if the patient becomes alkalotic. Regardless, treat the K levels immediately if they are dangerously high or low. And then seek and treat the underlying causes of acidaemia or alkalaemia.

Generally speaking – K levels of more than 2.8 mmols/L do not produce ECG changes. However, further decreasing levels will produce progressive changes on the ECG as described above. As with lots of other things, rapid, acute changes in electrolytes are often clinically more dangerous than slower, less acute changes.

Unfortunately, the ECG changes also do not correlate very well with the degree of hypokalaemia.

If you see ECG changes consistent with hypokalaemia, especially U waves and prolonged Qtc – you must keep the patient under close cardiac rhythm monitoring and ensure K levels are checked and if these are less than 2.8, start K replacement therapy otherwise the patient is at a real risk of a dangerous arrhythmia and potentially a cardiac arrest.

Always make sure that the patient is attached to a cardiac monitor when receiving IV potassium replacements. The consequences of too rapid rate of correction of hypokalaemia can be even more dangerous and potentially lethal.

Treatment of a prolonged QTc is basically that of the underlying cause if obvious which in this case would be urgent potassium replacement.

Note: Always consider and check for **hypomagnesemia** whenever treating significant hypokalaemia as the the two often co-exist. Lots of clinicians will recommend empirically replacing magnesium alongwith potassium replacement therapy in patients if magnesium levels are not immediately available.