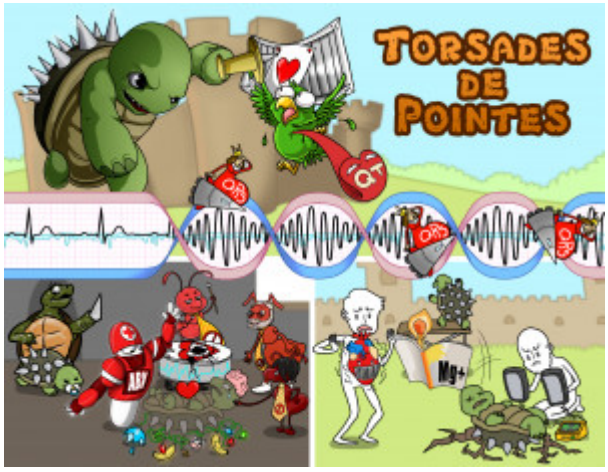
Torsades de Pointes

Torsades de pointes is a polymorphic ventricular tachycardia that occurs in the setting of QT interval prolongation. The name refers to the QRS appearing to twist around the isoelectric line. QT interval prolongation can be congenital or acquired. Acquired causes include antibiotics, antiarrhythmics, antipsychotics, antidepressants, and electrolyte deficiencies. Torsades can progress to ventricular fibrillation. Stable patients with torsades can be treated with magnesium sulfate, while unstable patients require defibrillation.



PLAY PICMONIC

Characteristics

Polymorphic Ventricular Tachycardia
Poly-morphing Vent Tac-heart-card
Torsades is a polymorphic ventricular tachycardia, meaning the ventricles will display a fast heart rate and each QRS will appear different from the next.

Prolonged QT Interval
Stretched QT-heart
The QT interval represents ventricular depolarization followed by repolarization. This interval can be increased, or prolonged, due to both congenital and acquired factors. Almost all medications and toxins which prolong the QT interval do so by inhibiting potassium channels in cardiac myocytes, preventing them from repolarizing properly. This increases the chance that an early afterdepolarization will occur during this time, leading to torsades.

QRS Twist Around Isoelectric Line
Queen's-Rocket-Ship Twisted Around Ice-electric Line
Torsades de pointes translates to “twisting of the points.” This refers to the EKG appearance where each QRS complex appears to cyclically move and twist around the isoelectric line, leading to its characterization as a polymorphic ventricular tachycardia.

Causes

Congenital
Present-from-birth
Long QT syndrome, which describes patients with a prolonged QT interval, can either be congenital or acquired. Congenital long QT syndromes are typically due to ion channel defects in cardiac myocytes, leading to a lifelong increased risk of torsades. Examples include Romano-Ward syndrome and Jervell & Lange-Nielsen syndrome.

Antibiotics
ABX-guy
There are many acquired causes of long QT syndrome, whereby a patient is exposed to a medication or toxin that prolongs the QT interval, increasing the risk of torsades. Certain antibiotics, especially macrolides (azithromycin, clarithromycin, etc) and fluoroquinolones (ciprofloxacin, levofloxacin, etc), are commonly implicated.

Antiarrhythmics
Ant-Tie-Arrhythmia-Drummer
Antiarrhythmics are medications which are given to suppress arrhythmias. Ironically, certain antiarrhythmics (class IA and III) can actually cause torsades due to their inhibitory effect on potassium channels in the heart, leading to a prolonged QT interval. Examples include amiodarone, sotalol and flecainide.

Antipsychotics

Ant-Tie-Psychiatrist

Antipsychotic medications inhibit dopamine receptors, but also inhibit a wide variety of other receptors and ion channels. In the heart, these effects can lead to a prolonged QT interval and potentially cause torsades. Examples include haloperidol, quetiapine, and risperidone.

Antidepressants

Ant-tie-depressed Emo

Certain antidepressants can have inhibitory effects on cardiac ion channels and also lead to torsades, especially if used in combination with other QT-prolonging medications. Examples include tricyclic antidepressants (eg., amitriptyline, doxepin) and selective serotonin reuptake inhibitors (eg., escitalopram).

Electrolyte Deficiencies

Electric-lights Broken

Electrolyte deficiencies are another important cause of torsades. Hypokalemia, hypomagnesemia and hypocalcemia are the most commonly implicated. Hypomagnesemia can directly cause hypokalemia. Patients taking diuretics or other medications which can cause electrolyte deficiencies as a side effect should be monitored closely.

Complications

Ventricular Fibrillation

Vent-heart Vibrating

While patients with brief episodes of torsades may present with typical arrhythmia symptoms like palpitations and lightheadedness, some patients will progress to a serious complication known as ventricular fibrillation. Ventricular fibrillation often presents with the patient suddenly collapsing due to an acute loss of cardiac function. On EKG, the ventricles will display a rapid, disorganized rhythm with loss of their normal contractions, leading to a lack of cardiac output.

Treatment

Stable: Magnesium Sulfate

Stable-ground Magnesium-magazine with Sulfur-match

For stable patients, meaning those with a stable blood pressure and no evidence of hemodynamic compromise, the treatment of choice for torsades is magnesium sulfate. This is given intravenously. Concurrently, any underlying causes should be corrected like electrolyte deficiencies or removal of offending agents.

Unstable: Defibrillation

Unstable-ground Defibrillator

For unstable patients with torsades, such as those with an unstable blood pressure, altered mental status, or other signs of hemodynamic compromise, the treatment is defibrillation. This uses an electric shock to attempt to convert the rhythm back to sinus rhythm.