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# Polymorphic Ventricular Tachycardia

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It was way back in late 1980s when I was doing my thesis work for Internal Medicine postgraduation that Prof. Ashokan Nambiar, my mentor, told me about polymorphic ventricular tachycardia with QT prolongation, better known as Torsades des Pointes. I was also given the opportunity to present four cases of Torsades des Pointes collected over 10 years for the State Conference of Cardiological Society of India. Those were the days when we did not have access Holter monitoring, leave alone implantable cardiac monitors, to document intermittent arrhythmia. In those days awareness about the arrhythmia was very low, unlike in current era when every resident in the Emergency Department is aware of polymorphic ventricular tachycardia.

It is important to recognize polymorphic ventricular tachycardia not only because it is potentially life threatening, but also because the management is different from the more common monomorphic ventricular tachycardia. Torsades de Pointes is a French phrase meaning "Twisting of the points." The name is because of the appearance of the ECG rhythm strip as illustrated in **Figure 1**. Sharp point of the QRS complexes shifts gradually from being positive to negative, as if the ECG strip is being twisted like a ribbon. Torsades de Pointes is often self-limited, but recurrent if the cause is not corrected. Prolonged episodes may need cardiopulmonary resuscitation and cardioversion as it can degenerate into ventricular fibrillation.

It was first described by Dessertenne F in 1966 as ventricular tachycardia with 2 variable opposing foci [1]. Important causes for torsades des pointes are electrolyte imbalances like hypokalemia and hypomagnesemia, and a wide variety of drugs. Important drug groups are non-sedating antihistamines, macrolides antibiotics, antifungals, antimalarials, tricyclic antidepressants, neuroleptics, and prokinetics [2]. Any new drug being developed, must undergo mandatory testing for QT interval prolongation before regulatory clearance. Congenital long QT syndromes are another rare cause of torsades des pointes. Acute ischemia and bradycardia can also predispose to QT prolongation and polymorphic ventricular tachycardia.

In case of drug induced torsades des pointes, the most important step is to avoid the offending drug. Correction of associated hypokalemia and hypomagnesemia are important. In bradycardic states like complete heart block, pacing will be useful in increasing the heart and reducing the QT interval which can suppress the arrhythmia. Intravenous magnesium sulphate is highly useful in most cases. Paradoxically, isoprenaline which itself can induce ventricular tachycardia, may be useful to increase heart rate and thereby reduce the QT interval and suppress torsades des pointes.

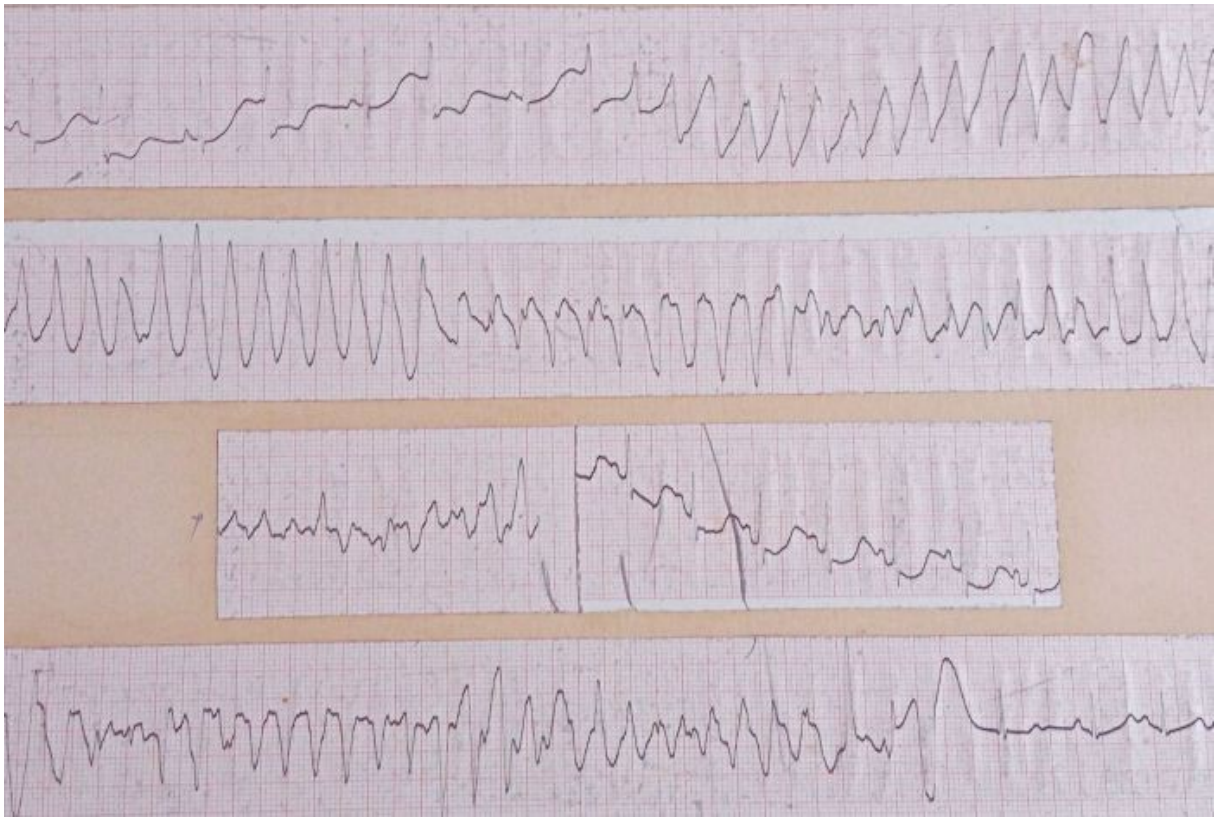

**Figure 1:** ECG showing polymorphic ventricular tachycardia

Suppression of torsades des pointes by intravenous magnesium is mediated through the blockade of L-type calcium channels [3]. Suppression can occur even without normalization of the prolonged QT interval. Magnesium is a physiological calcium antagonist within the myocardial cell. High levels of magnesium suppress calcium release from the sarcoplasmic reticulum and low levels of magnesium increases calcium release from the sarcoplasmic reticulum. Sarcoplasmic reticulum is an important intracellular store of calcium ions having important role in excitation contraction coupling in the myocardial cell. Intravenous magnesium has been used to suppress torsades des pointes induced by sotalol and it is presumed to be by suppression of early afterdepolarizations caused by sotalol [4].

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