**Introduction: Polymorphic Ventricular Tachycardia**

Polymorphic ventricular tachycardia (VT) is an unstable and potentially life-threatening heart rhythm disturbance. It involves rapid, irregular heartbeats that originate from the ventricles. Polymorphic VT is less common than monomorphic VT but is particularly concerning due to its association with sudden cardiac death. The most well-known type of polymorphic VT is torsades de pointes. Polymorphic VT occurs in the setting of underlying heart disease, electrolyte abnormalities, drug toxicity, or channelopathies that affect myocardial repolarization. Clinical pharmacists play a critical role in recognizing polymorphic VT on ECG, understanding the underlying mechanisms, and assisting with appropriate pharmacologic management. Prompt recognition and treatment of polymorphic VT are essential to prevent deterioration to ventricular fibrillation and cardiac arrest. This section will discuss the key features of polymorphic VT relevant for pharmacy professionals.

**Clinical Presentation**

Here is the clinical presentation section for polymorphic ventricular tachycardia:

* Symptoms associated with polymorphic VT include palpitations, lightheadedness, syncope, and chest pain. Hemodynamic instability with hypotension, altered mental status, and shock can occur.

* Polymorphic VT often presents with a rapid ventricular rate between 150-250 beats per minute.

* The ECG shows a irregular, continuously changing QRS morphology and axis - the complexes appear to twist around the isoelectric line.

* Prolongation of the QT interval is usually seen prior to initiation of polymorphic VT episodes.

* Risk factors include structural heart disease, electrolyte disturbances (hypokalemia, hypomagnesemia), drugs that prolong QT interval, bradycardia, pauses, congenital long QT syndromes.

* More common in older adults but can occur at any age. Slight male predominance.

* Sudden cardiac death can be the initial presentation in patients with undiagnosed congenital channelopathies.

* Syncope, cardiac arrest, or sudden death can occur if polymorphic VT deteriorates into ventricular fibrillation.

**Pathophysology**

Here is an expanded pathophysiology section for polymorphic ventricular tachycardia with additional paragraph explanations:

Polymorphic ventricular tachycardia arises from abnormal electrical conduction and repolarization within the ventricles. The changing QRS complexes represent a propagating wavefront through ventricular tissue that is heterogenous in its repolarization state.

* Polymorphic VT is often triggered by early afterdepolarizations that occur before completion of repolarization. Early afterdepolarizations are triggered by intracellular calcium overload and represent a form of abnormal automaticity.

* Conditions that promote early afterdepolarizations and initiation of polymorphic VT include:

* Structural heart disease like myocardial infarction or cardiomyopathy. These cause regional differences in repolarization due to scar or fibrotic tissue interspersed with surviving myocardial fibers. The heterogeneous repolarization creates dispersion of refractoriness.

* Bradycardia or acquired long QT interval. The increased time spent in repolarization allows the heterogeneity in Action Potential durations to manifest and become arrhythmogenic.

* Electrolyte disturbances like hypokalemia or hypomagnesemia. These directly impair myocardial repolarization through effects on ion channels like IKr.

* Drugs that block potassium channels like class IA and III antiarrhythmics. Delayed repolarization from potassium channel blockade also increases heterogeneity.

* Congenital channelopathies like long QT syndrome. Defective ion channels, especially reduced IKs current, facilitate early afterdepolarizations.

Once triggered by early afterdepolarizations, polymorphic VT is perpetuated by re-entry as the wavefront propagates through recovered and refractory tissue. The changing QRS vectors represent different areas of heterogeneity being recruited. If uncontrolled, polymorphic VT can degenerate into ventricular fibrillation and cause sudden cardiac death. Prompt recognition and treatment is essential.

**Diagnostic Approach**

A 12-lead ECG is the primary diagnostic tool and should be obtained immediately at symptom onset. It allows accurate diagnosis of polymorphic VT and provides clues to potential underlying causes.

Key findings on ECG include:

* Heart rate between 150-250 beats per minute
* QRS duration >120 ms
* Irregular, continuously changing QRS morphology and axis
* QT interval prolongation often precedes arrhythmia onset

Additional investigations are aimed at identifying triggers, ruling out reversible causes, and determining the underlying substrate:

* Serum electrolytes should be checked as hypokalemia and hypomagnesemia are common triggers for polymorphic VT. These require prompt correction.
* Cardiac enzymes like troponin evaluate for acute myocardial ischemia which can provoke polymorphic VT.
* A toxicology screen is important to rule out drug-induced QT prolongation as a trigger. Offending medications should be discontinued.
* Echocardiography assesses for structural heart disease like cardiomyopathy or valve disorders which provide substrate for reentry.
* A family history should be elicited given the potential for inherited channelopathies. Genetic testing in patients <40 years without structural disease can help diagnose conditions like long QT syndrome.
* In-hospital telemetry monitoring helps detect asymptomatic runs of polymorphic VT and analyze trends in QT interval and pauses.
* An electrophysiology study with programmed stimulation may induce polymorphic VT and provide insight on mechanism.
* Implantable loop recorders are useful for determining arrhythmia burden in cryptogenic cases.

In summary, the 12-lead ECG is the most important diagnostic tool. Additional testing aims to identify reversible triggers while ruling out underlying heart disease and inherited conditions. This guides appropriate management.

**Here is a comparison of monomorphic vs polymorphic ventricular tachycardia:**

Monomorphic and polymorphic ventricular tachycardias (VT) share some similarities but have key differences in presentation and management.

Similarities:

- Both originate from the ventricles

- Wide QRS complex (>120 ms)

- Potentially unstable depending on rate and duration

- Risk of deteriorating to ventricular fibrillation

Differences:

- Monomorphic VT has a regular rhythm with uniform QRS complexes suggesting a stable reentrant circuit

- Polymorphic VT has an irregular rhythm with continuously varying QRS suggesting changing activation

- Monomorphic VT is often due to scar from prior infarction

- Polymorphic VT is often due to acute ischemia, bradycardia, electrolyte imbalance, or medication effect

- Monomorphic VT management may involve IV antiarrhythmics, overdrive pacing, or catheter ablation

- Polymorphic VT management centers on identifying and correcting any trigger plus IV antiarrhythmics

- Monomorphic VT may be well tolerated if heart function is preserved

- Polymorphic VT is unstable by definition and requires prompt termination

- Monomorphic VT may be recurrent and require chronic management

- Polymorphic VT may be a one-time event if the trigger is eliminated

In summary, the regular vs irregular QRS complexes help distinguish monomorphic and polymorphic VT. Therapy differs based on the underlying mechanism and stability.

**Management - Overview**

The management of polymorphic ventricular tachycardia focuses on prompt termination of the arrhythmia to prevent deterioration into ventricular fibrillation and cardiac arrest. Intravenous antiarrhythmic medications like magnesium, amiodarone, or lidocaine are first-line for acute termination. Electrical cardioversion or defibrillation is used for medication-refractory cases.

Beyond arrhythmia termination, identifying and correcting any reversible triggers is a key component of management. This includes:

* Repleting electrolytes like potassium and magnesium for cases induced by hypokalemia or hypomagnesemia
* Removing any offending drugs that may prolong QT interval
* Treating any underlying bradycardia that may be contributing

Longer term, implantable cardioverter defibrillators (ICDs) are indicated for secondary prevention in survivors of cardiac arrest or recurrent polymorphic VT. Use of quinidine or catheter ablation helps manage refractory, recurrent cases. For congenital long QT syndrome, avoidance of QT prolonging drugs, restriction of strenuous activity, and family screening are all recommended.

In summary, management requires both acute termination and long-term prevention focused on reducing arrhythmia triggers and sudden death risk.

**Pharmacotherapy**

Electrical Cardioversion

* First-line for hemodynamically unstable VT
* Administer sedation beforehand if patient is conscious; short-acting agents like etomidate or propofol preferred
* Initial synchronized shock at 100 J or higher; subsequent shocks can increase energy up to maximum of 200 J if needed for conversion
* Higher energies may be required for conversion if poor ventricular function

Intravenous antiarrhythmic medications are preferred for stable polymorphic VT:

**Magnesium sulfate**

* is the drug of choice for torsades de pointes or polymorphic VT associated with long QT interval
* It corrects hypokalemia if present and shortens QT regardless of magnesium level.
* The dose is 2 g diluted in 10 mL D5W or normal saline administered IV over 5-20 minutes.
* This can be repeated up to 2 additional doses if needed.
* Monitor for hypotension.

Amiodarone

* Can be used first-line when polymorphic VT is not known to be long QT-related.
* Should be avoided if the QTc interval is markedly prolonged, as it can further prolong repolarization and worsen torsades de pointes.
* Give a 150  mg IV bolus over 20-60 minutes followed by an infusion of 1 mg/min for the first 6 hours then 0.5 mg/min.
* Monitor QTc interval, hypotension, and AV block. Stop amiodarone if QTc prolongs excessively.

Lidocaine

* is an alternative to amiodarone, especially if QTc prolongation is present.
* Give an IV bolus of 100-200 mg followed by an infusion at 1-4 mg/min titrated to suppress the arrhythmia.
* Monitor for CNS toxicity symptoms.

Overdrive pacing

* Done  with a temporary transvenous pacer can help suppress recurrent polymorphic VT episodes. Target a pacing rate of 90-110 bpm.
* Do not use pacing when QTc is markedly prolonged.

Isoproterenol infusion

* may be considered for polymorphic VT related to bradycardia or pauses.
* Titrate the infusion to keep the heart rate >70 bpm, typically starting at 2 mcg/min up to 10 mcg/min.
* Use with caution if long QT is present.

In summary, electrical cardioversion, IV antiarrhythmics like magnesium and amiodarone, and correction of reversible triggers comprise the mainstay of acute treatment for polymorphic VT in unstable patients.

The goal is to promptly terminate arrhythmia episodes and prevent deterioration into ventricular fibrillation.

**Tips for Board Exam Questions:**

* Recognize polymorphic VT is unstable and requires prompt termination with electrical cardioversion, IV magnesium, or amiodarone
* If a drug is mentioned, consider if it could prolong QT and predispose to polymorphic VT
* Recognize polymorphic VT has high risk of deteriorating to vfib and causing sudden death
* Focus on the key distinguishing features of polymorphic VT: irregular rhythm, changing QRS morphologies, association with QT prolongation

**Key Guidelines and Evidence**

**AHA/ACC/HRS (2017)**

* Intravenous magnesium can suppress episodes of torsades de pointes without necessarily shortening QT, even when serum magnesium is normal. Repeated doses may be needed, titrated to suppress ectopy and nonsustained VT episodes while precipitating factors are corrected.
* In patient with recurrent torases de pointes associated with acquired QT proklongation and bradycardia that cannot be suppressed with intravenous magnesium administration, increasing the heart rate with atrial or ventricular pacing or isoproterenol are recommended to suppress the arrhythmia
* Maintaining serum potassium between 4.5 mEq/L and 5 mEq/L shortens QT and may reduce the chance of recurrent torsades de pointes

**Key Study**

* Tzivoni D, Banai S, Schuger C, Benhorin J, Keren A, Gottlieb S, Stern S. Treatment of torsade de pointes with magnesium sulfate. Circulation. 1988 Feb;77(2):392-7.
  + Twelve consecutive patients who developed torsade de pointes (polymorphous ventricular tachycardia with marked QT prolongation, TdP) over a 4 year period were treated with intravenous injections of magnesium sulfate.
  + In nine of the patients a single bolus of 2 g completely abolished the TdP within 1 to 5 min, and in three others complete abolition of the TdP was achieved after a second bolus was given 5 to 15 min later.

**Clinical Scenarios**

**Scenario 1:**

A 67-year-old male with a history of coronary artery disease presents with gradual onset palpitations and lightheadedness. ECG shows a regular wide complex tachycardia at 170 bpm consistent with monomorphic VT. He receives IV amiodarone with successful restoration of normal sinus rhythm. One hour later, he develops recurrent tachycardia but now with irregular QRS complexes twisting around the baseline concerning for torsades de pointes.

This case highlights the importance of monitoring QTc interval after amiodarone administration and being alert for conversion to polymorphic VT. Amiodarone should be discontinued and IV magnesium given for recurrent torsades. Any electrolyte abnormalities must be corrected. Overdrive pacing can also help suppress episodes.

**Scenario 2**:

A 72-year-old female with a history of ischemic cardiomyopathy presents in polymorphic VT. She receives IV amiodarone but becomes more hypotensive. Repeat ECG shows QTc 550 ms.

This case illustrates amiodarone worsening hypotension and exacerbating torsades de pointes when baseline QTc is already markedly prolonged. Amiodarone should be stopped and IV magnesium given instead to shorten the QT interval. The patient may require temporary transvenous pacing if bradycardia or long pauses are present. Identifying and correcting any reversible QT prolonging triggers is key.

**Summary**

* Polymorphic VT is characterized by irregular, continuously changing QRS complexes, often with QT prolongation
* It is caused by heterogeneous repolarization that facilitates triggered activity and reentry
* Precipitants include electrolyte disturbances, medications, bradycardia, myocardial ischemia, and congenital channelopathies
* Diagnosis is by 12-lead ECG along with testing to identify reversible triggers
* IV antiarrhythmics like magnesium and amiodarone are first-line for acute termination
* Avoid amiodarone if QTc is markedly prolonged as it may worsen torsades de pointes
* Correct any reversible electrolyte, medication, or bradycardia triggers
* ICDs help provide backup protection against sudden death from recurrence
* Quinidine, ablation, or ICDs manage refractory cases

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