**Introduction: Monmorphic Ventricular Tachycardia**

Monomorphic ventricular tachycardia (MVT) is an abnormal heart rhythm characterized by fast, regular ventricular contractions originating from one ventricular focus. It is a significant concern as it can lead to hemodynamic instability, syncope, sudden cardiac death, or progress to ventricular fibrillation. MVT accounts for approximately 80% of ventricular tachycardias and affects around 100,000 people in the U.S. annually. It often indicates underlying heart disease like prior myocardial infarction, cardiomyopathy, arrhythmogenic right ventricular dysplasia, or long QT syndrome. Clinical pharmacists play a critical role in recognizing MVT on ECG, assisting with antiarrhythmic medication selection and administration, monitoring for effectiveness and adverse effects, and educating patients. Prompt recognition and appropriate pharmacotherapy are key to improving outcomes in MVT.

**Clinical Presentation**

Symptoms associated with MVT include:

* Palpitations, often described as pounding, racing, or fluttering
* Chest pain or tightness
* Dyspnea
* Presyncope or syncope
* Cardiac arrest

Signs on exam:

* achycardia with narrow QRS complex on ECG (rate usually 140-250 bpm)
* Hemodynamic instability - hypotension, acute heart failure

Associated risk factors and conditions:

* Structural heart disease - prior myocardial infarction, cardiomyopathy
* Electrolyte abnormalities - hypokalemia, hypomagnesemia
* Drugs that prolong QT interval
* Congenital syndromes - long QT syndrome, Brugada syndrome
* Men are more commonly affected than women
* Mean age of presentation is 65 years old

MVT can present across a spectrum from asymptomatic ectopy to unstable rhythms causing syncope, chest pain, shortness of breath, hypotension, or sudden cardiac death. It is crucial to obtain a 12-lead ECG, which will demonstrate a regular, monomorphic wide complex tachycardia. Hemodynamic instability warrants urgent electrical cardioversion.

**Pathophysology**

The pathophysiology underlying monomorphic ventricular tachycardia involves re-entry circuits within the ventricles, most often due to areas of scar tissue from prior myocardial infarction. Let's break this down:

* Normal cardiac electrical activity originates in the sinus node, propagates through the atria to the AV node, down the bundle branches, and depolarizes the ventricles from endocardium to epicardium.
* In MVT, abnormal automaticity occurs in ventricular tissue, disrupting this organized flow of electricity.
* MVT origins are often near the borders of infarcted tissue and healthy myocardium. The scar forms areas of slowed conduction, allowing re-entry circuits to form.
* This re-entry circuit allows the depolarization wavefront to travel in a continuous loop, causing a rapid, regular ventricular rhythm.
* As this abnormal focus is localized to one area, the QRS complexes generally appear uniform or "monomorphic" on ECG.
* Factors that increase automaticity like electrolyte disturbances or medications can trigger MVT episodes in susceptible individuals.

In summary, MVT arises from abnormal ventricular pacemakers and re-entry circuits, typically around areas of myocardial scar. The re-entry of the depolarization wavefront maintains the rapid, regular rhythm. Recognizing the underlying scar and triggers allows us to risk stratify patients and select appropriate management.

**Diagnostic Approach**

The diagnosis of monomorphic ventricular tachycardia relies on clinical presentation and ECG findings. Key diagnostic considerations include:

* Symptoms - Palpitations, chest pain, presyncope/syncope, and cardiac arrest increase suspicion for ventricular arrhythmia
* 12-lead ECG - Presence of a regular, monomorphic wide QRS complex tachycardia (>120 ms) at a rate typically 140-250 bpm
* Hemodynamic instability - Hypotension and acute heart failure may be present
* Underlying heart disease - History of prior MI, cardiomyopathy, long QT syndromes increases risk
* Laboratory data - Check electrolytes, cardiac biomarkers, toxicology screen
* Imaging - Echocardiography to assess structural heart disease and ventricular function
* Invasive electrophysiology study - Gold standard to define arrhythmia mechanism and location if ablation or ICD placement planned
* Differential diagnosis - SVT with aberrancy, preexcited AFib/flutter, PVCs, artifact

**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 monomorphic ventricular tachycardia centers around immediate stabilization and prevention of recurrence. Key principles include:

* Assess hemodynamic stability - unstable patients require urgent cardioversion
* Identify and correct reversible causes like electrolyte abnormalities
* Antiarrhythmic medications for rate control or rhythm conversion
* Catheter ablation for recurrent VT unresponsive to medications
* ICD placement for secondary prevention if high risk of sudden cardiac death
* Treatment of underlying heart disease like ischemic or nonischemic cardiomyopathy

The mainstay of acute management is electrical cardioversion for hemodynamically significant VT and intravenous antiarrhythmic medications for stable patients. Chronic management focuses on catheter ablation, ICD placement, and optimizing heart failure pharmacotherapy.

**Pharmacotherapy**

The pharmacologic management of monomorphic VT focuses on rapid restoration of sinus rhythm in unstable patients and preventing recurrence. Options include:

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

Amiodarone

* First-line for stable monomorphic VT along with procainamide
* IV/IO bolus 150 mg (50 mg/min) over 10 minutes
* Follow with infusion of 1 mg/min for first 6 hours (can use med port for compatibility), then 0.5 mg/min
* Oral maintenance dosing 200-400 mg daily
* Causes peripheral vasodilation, may worsen hypotension especially with rapid infusion
* Monitor thyroid function, liver enzymes, pulmonary toxicity
* Avoid in severe sinus node dysfunction due to bradycardia risk
* Interacts with warfarin, statins, antitubercular therapy

Procainamide

* IV/IO bolus of 10-15 mg/kg (1,000-1,500 mg) over 30-60 minutes, max 100 mg/min
* Stops infusion for hypotension or if VT terminates
* Can repeat bolus or start infusion at 1-4 mg/min if VT recurs
* Hypotension most common adverse effect, seen in up to 15%
* Monitor ECG for QT prolongation and widen QRS > 50% baseline
* Dose reduce for renal dysfunction; creatinine clearance <60 mL/min start at 7.5 mg/kg
* Avoid in heart block, myocardial depression, prolonged QT

Lidocaine

* IV bolus 1-1.5 mg/kg (or 100 mg) over 2 minutes
* Repeat bolus every 5 minutes until max of 3 mg/kg
* If VT recurs, start infusion at 1-4 mg/min
* Less negative inotropy than amiodarone or procainamide
* Monitor for CNS toxicity such as seizures, confusion
* Reduce dose in hepatic dysfunction
* Advantages over amiodarone and procainamide that is does routinely cause hypotension

Rationale for Guideline recommendations:

* Recent RCTs and meta-analyses show procainamide is more effective and safer than amiodarone for acute conversion of stable MVT but guidelines doesn’t favor one over the other.
* Amiodarone has higher rates of hypotension and other adverse cardiac effects.
* Lidocaine is less effective but offers an option with minimal cardiac effects.

**Tips for Board Exam Questions:**

1. Understand the indications for electrical cardioversion - first-line for unstable MVT. Know recommended starting energies and how to synchronize the device.
2. Be familiar with lidocaine as an alternative option with less negative inotropy compared to procainamide or amiodarone. Know lidocaine dosing, repeat dosing, infusion rates, and toxicity monitoring.
3. For questions on hypotensive patients, electrical cardioversion should be the go-to answer. Medications all have risks of worsening hypotension.

**Key Guidelines and Evidence**

Guideline Recommendations for Medications in Monomorphic VT

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| --- | --- |
| **Guideline** | **Recommendation** |
| AHA/ACC/HRS (2017) | * Procainamide (Class IIa) over amiodarone (Class IIb) for stable monomorphic VT |
| ESC (2015) | * Amiodarone recommended for stable monomorphic VT |

Summary of Key Evidence

* PROCAMIO Trial (2017): Procainamide superior to amiodarone for conversion of stable MVT (67% vs 38%, p<0.05) with fewer adverse events
* Marill et al (2010): Retrospective analysis found amiodarone and procainamide had similar efficacy for stable MVT (59% vs 43%, p=0.08)
* AHA/ACC/HRS guideline update (2017) changed recommendation to procainamide preferred over amiodarone based on efficacy and safety data

In summary, recent RCT and retrospective data support procainamide over amiodarone for acute, stable monomorphic VT. This led to an updated recommendation by AHA guidelines favoring procainamide.

**Clinical Scenarios**

**Scenario 1**

A 65-year-old male with a history of myocardial infarction presents with sudden onset of palpitations. His blood pressure is 92/64 mmHg, heart rate is 180 bpm, and ECG shows a regular, monomorphic wide complex tachycardia. He receives a procainamide infusion but after 20 minutes his blood pressure drops to 78/40 mmHg.

*This scenario demonstrates hypotension, a potential adverse effect of procainamide infusion. The recommended management would be to stop the infusion and proceed immediately to electrical cardioversion if blood pressure doesn’t rebound quickly.*

**Scenario 2**

A 55-year-old female with nonischemic cardiomyopathy has an implantable cardioverter defibrillator (ICD) in place. She presents with recurrent ICD shocks. ECG shows monomorphic VT at a rate of 210 bpm. She receives lidocaine 100 mg IV push but the rhythm persists.

*This scenario represents monomorphic VT unresponsive to lidocaine in a patient with an ICD. The next recommended step would be an amiodarone 150 mg IV bolus over 10 minutes followed by an infusion.*

**Summary**

* Monomorphic VT is characterized by a regular, monomorphic wide complex tachycardia, often occurring in structural heart disease
* Hemodynamically unstable patients warrant immediate electrical cardioversion
* For stable VT, procainamide is first-line based on recent evidence showing superiority over amiodarone
* Lidocaine offers an alternative with less negative inotropy but lower efficacy
* All medications should be paired with monitoring for effectiveness, recurrence of VT, and adverse events
* Correct reversible causes and consult electrophysiology for recurrent VT refractory to medications
* Clinical pharmacists play a vital role in appropriate antiarrhythmic selection, dosing, administration, and monitoring to optimize outcomes in monomorphic VT

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