

# Visual Diagnosis in Emergency Medicine



## NORMAL SINUS RHYTHM OBSCURED BY ARTIFACT

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### INTRODUCTION

The spectrum of ventricular tachycardia (VT) includes monomorphic and polymorphic VT. Torsades de pointes (TdP) is the specific polymorphic VT with associated long QTC interval. Presence of sinus rhythm or a QRS complex in even a single electrocardiogram (ECG) lead excludes TdP, but may not be readily apparent.

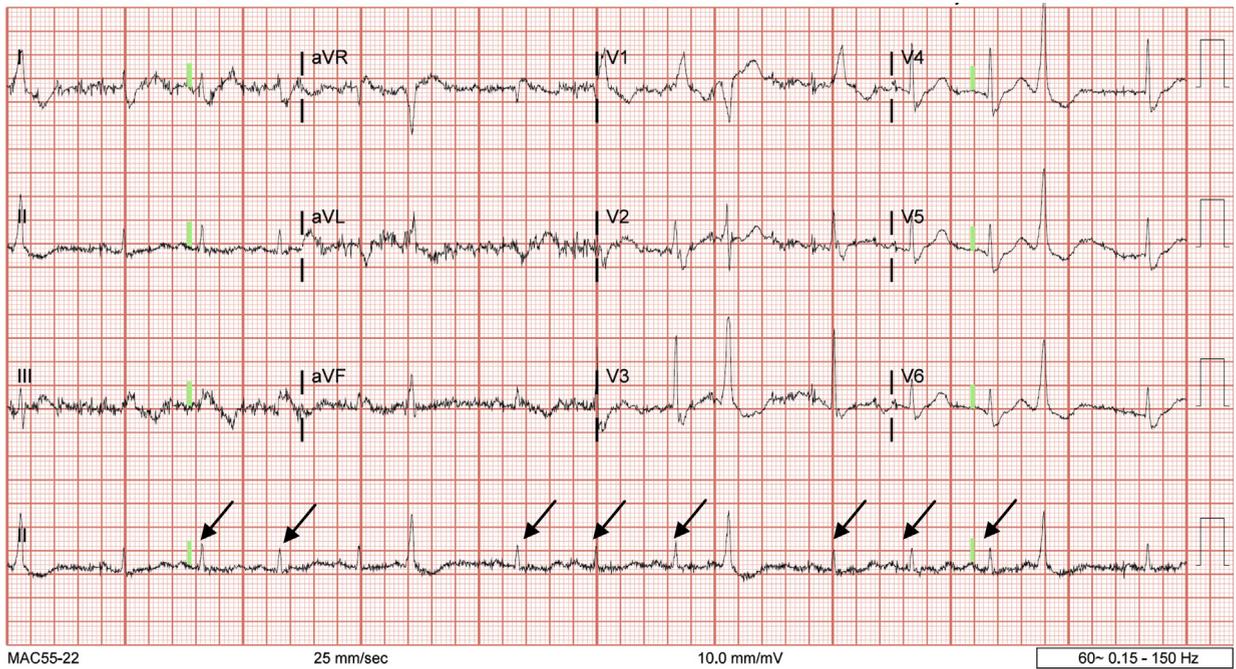
### CASE REPORT

A 90-year-old man with history of remote pacemaker placement, atrial fibrillation, coronary artery disease, and known bilateral lower extremity atherosclerosis presented to the Emergency Department with generalized weakness. He denied chest pain, palpitations, or shortness of breath, but did present with a resting hand tremor. The patient did not take any home medications. Initial blood pressure was 174/136 mm Hg with a heart rate of 92 beats/min, and narrow complex rhythm on the monitor. Triage ECG demonstrated tremor artifact with visible QRS complexes, occasional premature ventricular complexes (PVCs), QRS complex of 136 ms, and QTC of 525 ms (Figure 1). The patient's previous ECG from 3 years earlier demonstrated sinus rhythm with PVCs and a right bundle branch block with a QRS complex of 132 ms and QTC of 482 ms (Figure 2). Five minutes after arrival, the patient's apparent rhythm changed on the monitor and a repeat ECG was obtained (Figure 3). The

patient reported no chest pain, palpitations, or difficulty breathing. The patient was lying still, but had a continued resting hand tremor, unchanged from his initial presentation. Given stable vital signs and an asymptomatic patient, intravenous magnesium was administered for empiric treatment of possible TdP; repeat ECG was obtained afterward, which showed sinus rhythm with a right bundle branch block. Repeat blood pressure was 141/72 mm Hg with a heart rate of 85 beats/min. Potassium was 3.6 mmol/L (normal 3.5–5.1 mmol/L), and magnesium was 2.2 mg/dL (normal 1.7–2.4 mg/dL). Pacemaker interrogation showed sinus rhythm with no events. No further ECGs were obtained during the patient's hospital admission.

### DISCUSSION

This case initially raised concern for TdP, but there are several clues that the underlying rhythm is normal sinus with artifact. Re-evaluation of the ECG at the time of apparent rhythm change demonstrated normal sinus rhythm in lead III (Figure 3), which effectively ruled out polymorphic VT or TdP. The presence of discernable QRS complexes demonstrated in Figure 4 also verify that the patient's underlying rhythm was indeed normal sinus. Closer inspection demonstrates that the QRS complexes present on the patient's initial ECG (Figure 1) align with the discernable QRS complexes present on the ECG at the time the patient's apparent rhythm changed



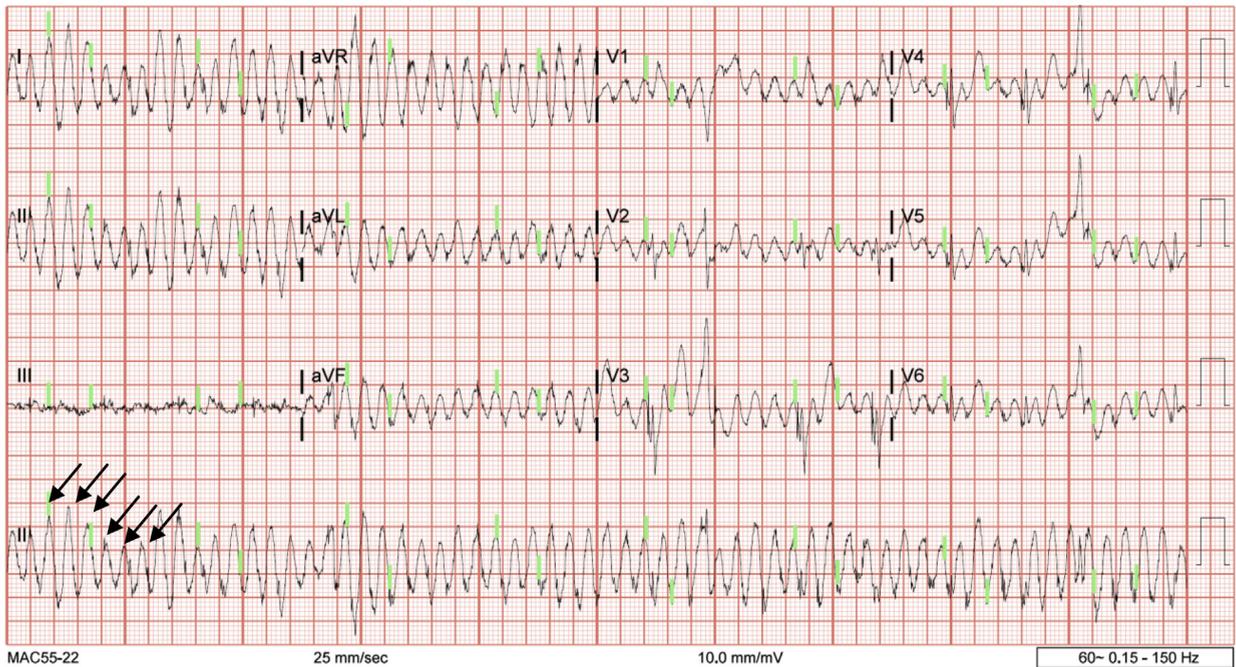
**Figure 1. Triage electrocardiogram demonstrating motion artifact with visible QRS complexes, occasional premature ventricular complexes, QRS complex of 136 ms, and QTc 525 ms.**

(Figure 2), as demonstrated with the superimposed rhythm in Figure 4. Thus, the presence of sinus rhythm or discernable QRS complexes in any single lead rule out the potential for TdP.

The patient presented with generalized weakness and a resting hand tremor. The initial ECG (Figure 1) demonstrated long QTc and PVCs; the abrupt apparent rhythm change to a wide complex tachycardia with variable



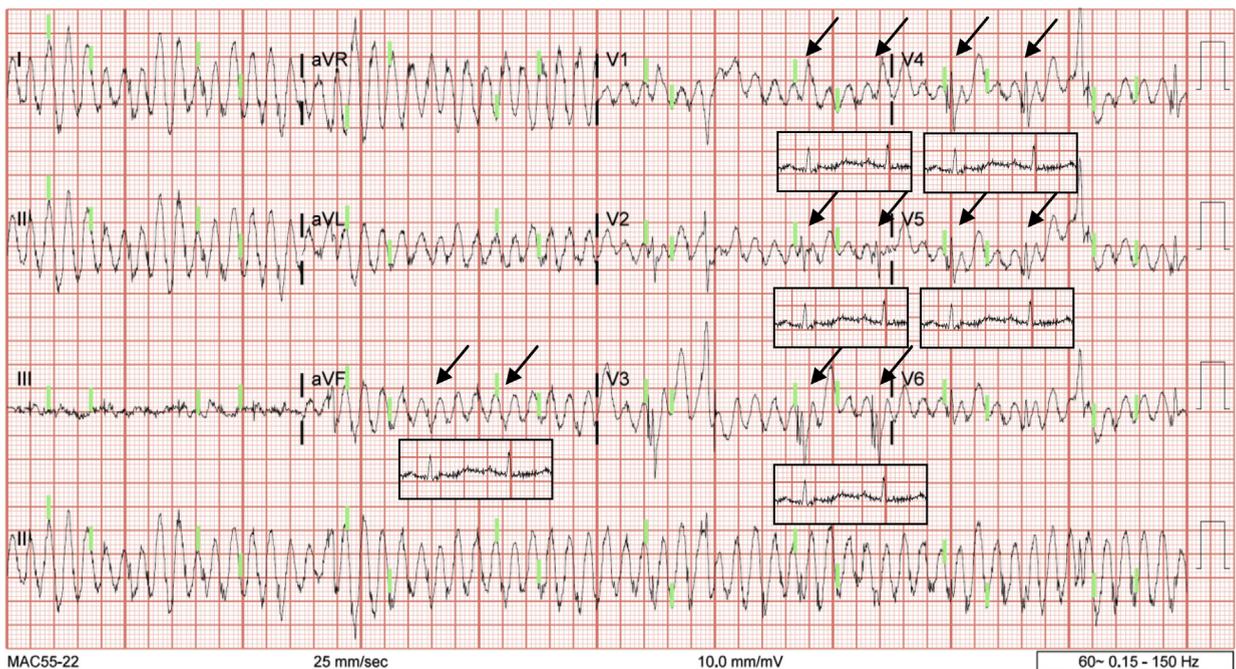
**Figure 2. Old electrocardiogram demonstrating sinus rhythm with premature ventricular complexes, right bundle branch block, QRS complex of 132 ms and QTc of 482 ms.**



**Figure 3. Electrocardiogram (ECG) concerning for rapid, wide complex tachycardia. There is beat-to-beat variation of the QRS complex width and amplitude. These findings were concerning for polymorphic ventricular tachycardia or torsades de pointes, especially given the long QTC interval present on the triage ECG.**

morphology QRS complexes (Figure 3) was concerning for polymorphic VT or TdP, especially because long QTC with presence of PVCs often instigates TdP, and the patient’s initial QTC was 525 ms. The QTC interval

is considered prolonged if >430 ms in males and >450 ms in females (1). Because TdP is a life-threatening rhythm that can result in sudden hemodynamic collapse and frequently degenerates into ventricular



**Figure 4. Underlying rhythm is normal sinus rhythm. QRS complexes are indicated by arrows. These QRS complexes are identical to the rate of the QRS complexes present on the patient’s initial electrocardiogram (black rectangle).**

fibrillation, immediate recognition and management is essential. The QRS morphology in lead III, axis, and interval duration remained unchanged in the triage ECG, compared with the ECG with concern for possible TdP, which strongly suggests that polymorphic VT was not present and the findings in the other leads represented artifact. Although we do not know why this artifactual finding was observed, the apparent rhythm change is suspected to be from artifact from the patient's resting tremor; it is unlikely the artifact was caused by a machine malfunction, as the patient's repeat ECG in [Figure 4](#) and subsequent ECGs showed no signs of similar artifact. The presence of sinus rhythm or a QRS complex in even a single lead definitively excludes the diagnosis of both VT and TdP. Management

options for an unstable patient in TdP include immediate defibrillation, whereas 2 g of intravenous magnesium sulfate can be administered over 3–5 min in stable patients, and electrolyte derangements—especially magnesium and potassium—should be corrected, and overdrive pacing or isoproterenol infusions, which increase the resting heart rate, should be considered (2).

## REFERENCES

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2. Roden DM. A practical approach to torsade de pointes. *Clin Cardiol* 1997;20:285–90.