A 66-year-old woman presented to an emergency department late one afternoon with a 3-day history of diffuse abdominal pain, inability to eat, nausea, vomiting, diarrhea, and persistent coughing. In the triage area she rated her abdominal pain as being 6, on a 10-point pain scale. Her initial vital signs were all unremarkable. The woman’s medical history was significant for type II diabetes mellitus, renal failure (for which she was undergoing dialysis 3 times a week, with her last dialysis session 2 days earlier), amputation of her left leg above the knee, and amputation of her right leg below the knee. In addition to her obvious comorbidities, the patient’s current drug list (Table 1) suggested a history of hypercholesterolemia, hypertension, hypothyroidism, nonspecific cardiac disease, and depression.

A medical screening examination was conducted and 8 mg of orally disintegrating ondansetron (Zofran) was administered for persistent nausea and vomiting. A 12-lead electrocardiogram (ECG) completed at triage (Figure 1) was remarkable for left ventricular hypertrophy and QT interval prolongation. After the drug was administered and electrocardiography was performed, the patient was returned to the waiting room to await further treatment.

Shortly thereafter, the patient self-discharged from the emergency department before receiving definitive treatment. Upon making a follow-up phone call, it was discovered that the patient had been found unresponsive in bed approximately 4 hours after leaving the emergency department. She was pronounced dead at the scene by emergency care providers. Because of her extensive medical history, the woman’s family declined an autopsy, and her primary physician attributed the death to complications of diabetes mellitus, end-stage renal disease, and hypertension.

**Discussion**

Without definitive electrocardiographic evidence or autopsy confirmation, the cause of death cannot be determined. However, upon review of this case for evidence of her impending death, multiple risk factors for torsades de pointes (pronounced tor-sod’ day pwan’) become evident (Table 2). Alone, each risk factor could be considered benign. However, in combination, they form a “perfect storm” scenario for this lethal cardiac dysrhythmia.

**TORSADES DE POINTES**

Torsades de pointes (also known as torsades) is defined as a polymorphic ventricular tachycardia that is preceded by a prolonged QT interval, as noted on an ECG. The QT interval represents ventricular repolarization, and the interval may increase or decrease in response to external stimuli. As opposed to the rare congenital form of torsades, acquired cases of QT interval prolongation are relatively common. A recent study of critical care patients found that nearly one quarter of all patients (n = 1039) experienced QT interval prolongation greater than 500 milliseconds at some point during their hospitalization.

In 2010 a practice standard entitled “Prevention of Torsades de Pointes in Hospital Settings” was jointly released by the American Heart Association and the American College of Cardiology. This practice standard built on an earlier standard that recommended QT interval monitoring for at-risk patients. At-risk patients were defined as those (1) receiving proarrhythmic drugs, (2) having electrolyte abnormalities (hypokalemia, hypomagnesemia), or (3) experiencing bradydysrhythmias.
The aforementioned study found that 69% of critical care patients (n = 1039) had one or more of these monitoring indications.

The most important contributor to acquired QT interval prolongation is the use of certain cardiac and noncardiac medications. Proarrhythmic drugs commonly

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**TABLE 1**  
**Current drugs taken by the patient**

- Aspirin
- Cyclobenzaprine<sup>a</sup>
- Insulin
- Levothyroxine
- Metoprolol
- Nephro-Vite
- Paroxetine (Paxil) (conditional risk for torsades<sup>a</sup>)
- Prinivil
- Renagel
- Simvastatin
- Amlodipine
- Ondansetron (Zofran)<sup>b</sup> (possible risk for torsades<sup>b</sup>)

<sup>a</sup>Potentially proarrhythmic drugs as classified by the Arizona Center for Education and Research on Therapeutics (www.qtdrugs.org).

<sup>b</sup>Given during emergency department visit.

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**TABLE 2**  
**Common risk factors for torsades de pointes<sup>1</sup>**

- Female gender<sup>a</sup>
- Hypokalemia<sup>a</sup>
- Bradycardia
- Congestive heart failure
- Digitalis therapy
- Proarrhythmic drug (polypharmacy)<sup>a</sup>
- Left ventricular hypertrophy<sup>a</sup>
- Congenital long QT syndrome
- Hypomagnesemia
- Heart disease<sup>a</sup>
- Renal failure<sup>a</sup>
- Hypothyroidism<sup>a</sup>

<sup>a</sup>Risk factors present in case study.

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**FIGURE 1**  
A 12-lead electrocardiogram of a 66-year-old woman: 58 beats per minute, normal axis—no deviation, left ventricular hypertrophy (Sokolow-Lyon criteria: sum of S in V1 plus R in V5 or V6 > 35 mm), nonspecific T-wave abnormalities and QT/QTC interval prolongation (548 msec/538 msec, respectively).

The aforementioned study found that 69% of critical care patients (n = 1039) had one or more of these monitoring indications.
act by directly blocking the outflow of potassium repolarizing currents within the cardiac cells or by interacting with another drug to potentiate the second drug’s proarrhythmic effects. When cardiac repolarization time increases (signified by QT interval prolongation), cardiac cells are vulnerable to dysrhythmia development. An impulse during this critical phase can trigger an episode of torsades. Such episodes may be either self-limiting or lethal.

**CLINICAL IMPLICATIONS**

Any patient with a QT interval greater than 500 milliseconds requires close observation and serial QT interval measurement. Measurements should be obtained using a consistent lead that has an easily identifiable QRS complex and T wave onset and offsets. Because the QT interval shortens with fast heart rates and lengthens at slow heart rates, the modifying effect of heart rate on the QT interval should be accounted for by using an established correction formula. Two of the methods most commonly used in clinical practice are the Bazett formula ($QT_c = QT/RR^{1/2}$) and the Fridericia formula ($QT_c = QT/RR^{1/3}$). Electrocardiographic signs of impending torsades include T wave alternans, polymorphic premature ventricular contractions, long pauses, noncerebral U waves, and unsustained torsades.

In addition to vigilant patient monitoring, administration of known proarrhythmic drugs should be halted (Class I, Level A recommendation) (for evidence rating, see Table 3). Electrolyte levels (specifically potassium and magnesium) should be maintained within normal limits (Class IIb, Level B recommendation). Patients with torsades also may benefit from supplemental magnesium (Class IIa, Level B recommendation), even in the presence of normal serum magnesium levels. Pacing is warranted in patients with symptomatic bradydysrhythmias who are experiencing episodes of torsades (Class I, Level A recommendation).

**Conclusion**

Although cause of death cannot be definitively established, our 66-year-old sudden death patient had multiple torsades risk factors. Torsades in the hospital setting is potentially preventable with adequate QT interval surveillance, identification of at-risk patients, removal of proarrhythmic drugs, and correction of abnormal electrolyte levels.

**Addendum**

On September 15, 2011, the Food and Drug Administration (FDA) released a safety communication stating that abnormal heart rhythms may be associated with the use of Zofran (Ondansetron). Recommendations for labeling changes have been made and a safety review is currently ongoing.

**REFERENCES**


This section features actual emergency situations with particular educational value for the emergency nurse. Contributions (3 to 5 typed, double-spaced pages) should include a case summary focused on the emergency care phase, accompanied by pertinent case commentary. Submissions to this column are encouraged and may be sent to Laura M. Criddle, PhD, RN, CEN, CCNS, FAEN http://ees.elsevier.com/jen