Implantable Cardioverter Defibrillator (ICD) for Polymorphic Ventricular Tachycardia (VT) Due to Coronary Vasospasm

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We report a case of a young woman who presented with atypical angina. During an episode of chest pain she had a documented run of sustained polymorphic ventricular tachycardia (VT). In addition to medical therapy, she received an ICD to prevent future episodes of sudden cardiac death. Journal of Hospital Medicine 2009;4:E23–E25. © 2009 Society of Hospital Medicine.

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Arrhythmias are well-described in patients with vasospastic angina. Coronary vasospasm may occur in the setting of angiographically normal or diseased coronary arteries. Patients with vasospastic angina are at increased risk of sudden death. However, it is unclear which of these patients would benefit from implantable cardioverter defibrillator (ICD) insertion.

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Case Report

A 38-year-old woman was admitted with an episode of severe central chest pain. The pain was sharp, localized, occurred at rest, and resolved spontaneously after about 2 hours. She reported intermittent but shorter episodes of similar chest pain for the preceding 5 months. These episodes were associated with palpitation and lightheadedness. She was a smoker with past medical history of hyperlipidemia, asthma, and psoriasis. There was no family history of sudden cardiac death or premature coronary artery disease. On presentation, she was pain-free, and vitals were stable. Electrocardiogram revealed normal sinus rhythm, no ischemic ST segment changes, and a QTc interval of 479 msec. Serum troponins were normal, serum potassium was 3.5 mmol/L, and serum magnesium was 1.9 mg/dL. While she was being monitored on telemetry, she suddenly experienced chest pain with palpitation. The telemetry recording of the event showed transient ST segment elevation followed by an episode of sustained polymorphic ventricular tachycardia (VT) (Figure 1). She remained hemodynamically stable and did not lose consciousness. The VT was self-terminating.

Coronary angiogram revealed moderate 2-vessel disease and spontaneous spasm of the dominant left circumflex artery. Therapy was initiated using both an oral nitrate and calcium channel antagonist. Potassium and magnesium levels were corrected with supplementation. On further questioning, she reported 1 episode of near-syncpe in the past. In view of the above history and a documented episode of spontaneous sustained polymorphic VT, an implantable cardioverter defibrillator (ICD) was implanted. She was strongly advised to quit smoking and was discharged home in stable condition. Three months later, she was admitted with recurrence of similar episodes of chest pain and dizziness, and multiple shocks from her ICD. Interrogation of the ICD revealed 5 episodes of polymorphic VT that were appropriately terminated with ICD discharges. The doses of calcium channel antagonist and oral nitrate were maximized, and she was discharged home in stable condition.

Discussion

Our case highlights an important management dilemma in patients with vasospastic angina. ICD implantation in this group has been reported in patients resuscitated from cardiac arrest. Our patient was recognized to be at high risk of sudden death but had never experienced cardiac arrest.

An increased incidence of sudden cardiac death, VT, and ventricular fibrillation has been observed during episodes of vasospastic angina. In a retrospective multicenter study of 349 patients with vasospastic angina, VT or ventricular fibrillation was noted in 6.5% of patients.1 Sudden death was reported in 2% of the patients (mean follow-up period, 3.4 years), of whom the majority had ST segment elevation during anginal attacks. Increased ventricular vulnerability has been noted even during symptom-free periods.2 Some cases of unexplained out-of-hospital cardiac arrest and sudden deaths may be secondary to coronary artery spasm.3 In a prospective study of 356 survivors of out-of-hospital cardiac arrest, increased ventricular vulnerability was noted even during symptom-free periods.4
FIGURE 1. Telemetry recording demonstrating ST segment elevation followed by onset of polymorphic ventricular tachycardia. Arrows denote ST segment elevation typically associated with an attack of vasospastic angina.

Conclusions

In summary, patients with vasospastic angina are at increased risk of sudden death, especially during an episode of angina. Some cases of unexplained sudden death and malignant ventricular arrhythmia are probably a consequence of acute myocardial ischemia resulting from coronary arterial spasm. Early recognition and treatment of polymorphic VT is critical in preventing sudden cardiac death. In the absence of myocardial infarction, ST segment elevation preceding an episode of syncope or arrhythmia should raise the suspicion of coronary vasospasm as the underlying etiology. ICD placement is potentially beneficial in patients with coronary spasm who are at high risk of sudden cardiac death. Larger trials with longer follow-up periods would help clinicians make this decision with greater confidence.

References

7. Sugishii M, Takatsu F. Cigarette smoking is a major risk factor for coronary spasm. Circulation. 1993;87:76–79.

researchers have reported a strong association between cigarette smoking and coronary spasm. Patients with known or suspected coronary artery spasm should be strongly discouraged from smoking. Our patient, in addition to being a smoker, had 2 of the 3 risk factors described by McAlpin, namely, documented serious arrhythmia and absence of high-grade coronary stenosis. Considering these risk factors, an ICD was implanted. To our knowledge this is the first reported case of ICD insertion in a patient with vasospasm-induced VT who had never experienced cardiac arrest.

artery spasm who had silent ischemic events associated with life-threatening ventricular arrhythmias. Interestingly, in 2 of the 5 patients, onset of ventricular arrhythmia correlated with reperfusion, rather than ischemia.

Calcium-channel antagonists and nitrates are accepted as the first-line treatment for vasospastic angina. Although this therapy improves prognosis, the risk of ventricular arrhythmia and sudden death is not eliminated. The data regarding use of ICDs in patients with coronary vasospasm are limited to case reports. Lacroix et al. reported 2 patients with vasospastic angina resuscitated from out-of-hospital cardiac arrest who received ICDs. Postimplantation, at 4 months and 11 months, respectively, each of the 2 patients had appropriate ICD discharges. Fuertes et al. reported a patient resuscitated from cardiac arrest due to ventricular fibrillation related to an episode of angina. The patient had vasospasm despite intensive medical therapy and had an ICD implanted. The above previously published cases describe ICD implantation for patients resuscitated from cardiac arrest.

However, in patients with coronary vasospasm who have never experienced cardiac arrest, it is unclear which subset would benefit from an ICD. Electrophysiological studies are not always helpful in identifying these patients. In the study by Myerburg et al., only 1 out of the 5 patients with coronary artery spasm had inducible arrhythmia during electrophysiological testing. Clinical features associated with increased risk of sudden death were reported by McAlpin in a study of 81 patients with vasospastic angina. The risk of sudden death was tripled by the presence of either a history of angina-linked syncope or documentation of serious arrhythmia complicating attacks. Paradoxically, the risk was increased by the absence of high-grade coronary artery stenosis. Some