*Case Reports*

# Ventricular Tachycardia Associated with Radiation-Induced Cardiac Sarcoma

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*Cardiac tumors can lead to distinct electrocardiographic changes and ventricular arrhythmias. Benign and malignant cardiac tumors have been associated with ventricular tachycardia. When possible, benign tumors should be resected when ventricular arrhythmias are intractable. Chemotherapy can shrink malignant tumors and eliminate arrhythmias.*

*We report the case of a 52-year-old woman with breast sarcoma whom we diagnosed with myocardial metastasis after she presented with palpitations. The initial electrocardiogram revealed sinus rhythm with new right bundle branch block and ST-segment elevation in the anterior precordial leads. During telemetry, hemodynamically stable, sustained ventricular tachycardia with right ventricular localization was detected. Images showed a myocardial mass in the right ventricular free wall. Amiodarone suppressed the arrhythmia.*

*To our knowledge, this is the first report of ventricular tachycardia associated with radiation-induced undifferentiated sarcoma. We discuss the distinct electrocardiographic changes and ventricular arrhythmias that can be associated with cardiac tumors, and we review the relevant medical literature.* ***(Tex Heart Inst J 2014;41(6):620-5)***

***Key words:*** *Amiodarone/administration & dosage/adverse effects/therapeutic use; arrhythmias, cardiac/diagnosis; heart neoplasms/complications/radiotherapy/secondary; neoplasms, radiation-induced/therapy; tachycardia, ventricular/diagnosis/drug therapy/etiology/physiopathology/prevention & control*

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Metastatic cardiac tumors can be diagnosed after various presentations, such as ventricular arrhythmia. We describe the case of a woman who had breast cancer with resultant radiation-induced sarcoma. Myocardial invasion was confirmed after she presented with ventricular tachycardia (VT). In addition, we review tumor-associated VT and the relevant medical literature.

## Case Report

In July 2012, a 52-year-old woman presented with palpitations. In 2001, she had been diagnosed with left-breast cancer and had undergone lumpectomy, radiation, and chemotherapy with adriamycin, cytoxan, and taxol. Because of a reported history of cardiomyopathy, she had not been given herceptin. In December 2010, she was diagnosed with radiation-induced left-breast sarcoma with extension to the pectoralis muscles and lungs. A preoperative echocardiogram revealed a left ventricular ejection fraction (LVEF) of 0.45. The patient underwent tumor resection and neoadjuvant chemotherapy with 3 cycles of etoposide and ifosfamide, then declined further chemotherapy. Her LVEF decreased to 0.30 in September 2011, and angiotensin-converting enzyme inhibitor and β-blocker therapy was initiated. In November 2011, computed tomograms revealed an interval increase in lung nodules. The patient underwent resection and 2 cycles of chemotherapy with gemcitabine and taxotere, then declined additional chemotherapy. In March 2012, an echocardiogram revealed an LVEF of 0.20 to 0.25. Because of her poor prognosis after refusing chemotherapy, she was not offered a defibrillator.

At the July 2012 presentation, the results of physical examination were unrevealing. The results of laboratory tests, including electrolytes and cardiac enzymes, were within normal limits. An electrocardiogram (ECG) on presentation revealed sinus rhythm with right bundle branch block (RBBB) and ST-segment elevation in leads $V_1$ through $V_3$ (Fig. 1A)—new since an ECG a year earlier (Fig. 1B). An echocardiogram revealed a new large, fixed echogenic mass on the right ventricular (RV) free wall with protrusion into the RV cavity (Fig. 2). In the cardiac care unit, the patient developed sustained wide-complex tachycardia that was consistent with VT (Fig. 3). The VT had

a cycle length of 440 ms and left bundle branch block morphology with a left superior axis, consistent with origination from the anatomic location of the cardiac tumor. The VT recurred despite intravenous metoprolol therapy, so intravenous amiodarone was initiated.

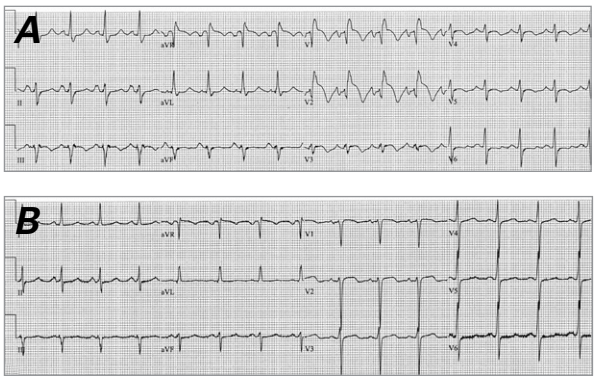


***Fig. 1*** ***A****) Presenting electrocardiogram shows sinus rhythm with right bundle branch block and ST-segment elevation in leads $V_1$ through $V_3$, suggesting tumor infiltration of the myocardium.* ***B****) Baseline electrocardiogram from one year earlier shows only sinus rhythm with voltage criteria for left ventricular hypertrophy.*

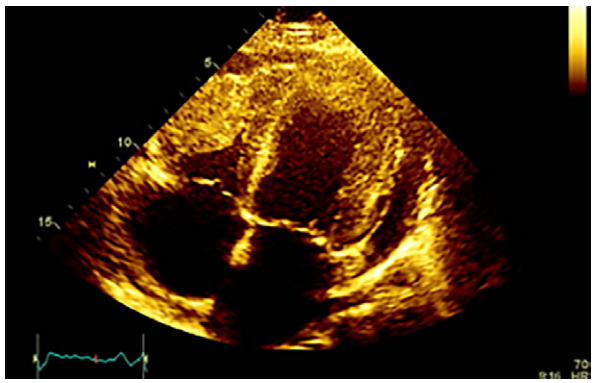

***Fig. 2*** *Transthoracic echocardiogram (apical 4-chamber view) reveals a large mass in the right ventricular free wall and a moderate-sized pericardial effusion.*

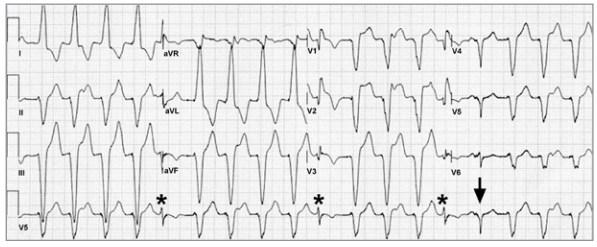

***Fig. 3*** *Twelve-lead electrocardiogram shows ventricular tachycardia and left bundle branch block morphology with a superior and leftward axis, consistent with a right ventricular origination of the tumor. Capture beats (asterisks) and a fusion beat (arrow) are noted.*

A computed tomogram showed an interval increase in pulmonary metastases. Cardiac magnetic resonance images revealed a 5.6 × 2.9-cm sessile mass in the RV free wall; peripherally delayed enhancement was consistent with metastasis of the sarcoma to the heart (Fig. 4). The intravenous amiodarone suppressed further runs of sustained VT; however, acute hepatotoxicity developed after one day (peak values of bilirubin, 5.1 mg/dL; aspartate aminotransferase, 6,053 U/L; alanine aminotransferase, 2,841 U/L; and alkaline phosphatase, 161 U/L). When amiodarone was discontinued, the liver enzyme values resolved. However, the VT recurred with shorter cycle lengths (Fig. 5); this and the patient's hemodynamic instability necessitated external defibrillation. Oral amiodarone suppressed the VT and caused no elevation in the liver enzyme levels.[1] Defibrillator implantation and catheter ablation were considered; however, because of the patient's poor overall prognosis, these therapies were thought to be inappropriate.

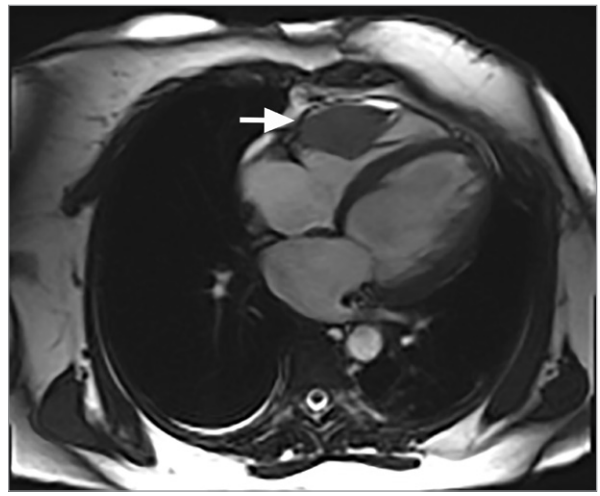

***Fig. 4*** *Cardiac magnetic resonance image shows a 5.6 × 2.9-cm sessile mass in the right ventricular free wall. Peripherally delayed enhancement is consistent with metastasis of the sarcoma to the heart (arrow).*

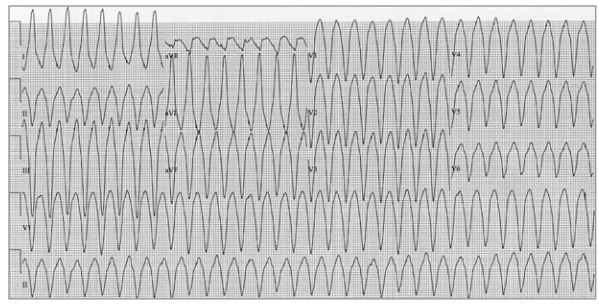

***Fig. 5*** *After intravenous amiodarone was discontinued, electrocardiogram shows recurrent ventricular tachycardia with a faster cycle length than before (320 ms) and morphology similar to that in the previous electrocardiogram (see Fig. 3).*

Before the patient was discharged from the hospital, she was fitted with a LifeVest® portable external defibrillator (Zoll Medical Corporation; Pittsburgh, Pa) for secondary prevention. She resumed chemotherapy for the metastatic sarcoma. During 6 months of follow-up, she had no recurrences of VT. However, the metastatic cancer progressed, and the patient entered hospice care and died in February 2013.

## Discussion

Cardiac tumors arise from a primary neoplasm, from direct invasion of the heart by a neighboring tumor, or through metastasis. A 1.5%-to-20% prevalence of cardiac metastases has been found in autopsy series of cancer patients.[2,3] Most cardiac sarcomas are secondary to metastasis; primary cardiac sarcomas occur in only 1 in 10,000 people in the general population.[4] The incidence of radiation-induced sarcoma of the breast is approximately 0.2% to 0.3% at 10 years; the 5-year survival rate is 36%.[5]

### Electrocardiographic Changes Associated with Cardiac Tumors

In cases of myocardial metastases, the ECG might be unchanged or might display a variety of findings, including ST-T changes, conduction disturbances, and arrhythmias. In our patient, the cardiac involvement of the sarcoma was associated with substantial ECG changes, including RBBB and marked ST-segment elevation in the anterior precordial leads. The presence of RBBB can suggest but does not necessarily predict tumor infiltration of the RV.[6] In one series of patients with myocardial metastases, an ECG pattern of myocardial injury or ischemia (ST-segment elevation or T-wave inversion, respectively) was highly specific (96%) but not sensitive (40%) for establishing cardiac infiltration.[7] In another series of patients with echocardiographically confirmed myocardial infiltration, ST-segment elevations were both sensitive (77%) and specific (89%) in detecting cardiac involvement.[6] Other ECG changes associated with cardiac metastases are atrial arrhythmias and low voltage.[7] Therefore, it is reasonable to use ECG as a screening tool for myocardial infiltration, while applying appropriate discretion in those patients who might also be at risk for common forms of cardiovascular disease. In a cancer patient whose primary tumor might metastasize to the heart, conduction abnormalities or new ST-segment changes warrant the use of diagnostic echocardiography, computed tomography, or cardiac magnetic resonance.

Proposed pathophysiologic mechanisms for ST-T changes in patients with cardiac metastases include myocardial ischemia caused by the tumor's compressing the coronary arteries, and direct extension of the tumor into the lumina of the coronary arteries (including the possible embolization of tumor fragments). Tumor invasion into the pericardium can result in pericarditis with secondary ST-T changes. Pericardial effusion might result in low QRS voltage or electrical alternans. Convex ST elevation, as in our patient, is most likely caused by continuous myocardial injury from direct pressure or physiochemical action.[7]

### Tumor-Induced Ventricular Tachycardia

Ventricular arrhythmia can be the initial presentation of a primary cardiac tumor or cardiac metastasis. The ECG-determined location of VT often correlates with the anatomic location of the cardiac tumor.[8] The mechanism of ventricular arrhythmias associated with cardiac tumors has not been well characterized. In an early report of VT in a patient who had metastatic melanoma in the posterior left ventricle,[9] the authors postulated 3 possible mechanisms of the VT. First, macro-reentry might be present around the base of the tumor. Second, altered myocardial architecture might lead to dispersed repolarization, anisotropic conduction, and micro-reentry. Third, humoral elements released from myocardial cells or the direct compression of myocardial fibers might lead to automatic ventricular arrhythmias.[9] Electrophysiologic studies have supported each of these proposed mechanisms. Reentry has been established by entrainment during tachycardia.[10] In a report of VT and primary cardiac fibroma,[11] triggered activity was the proposed mechanism. Burst pacing induced that patient's VT, whereas programmed stimulation did not. Activation mapping revealed radial activation away from the fibroma, without signs of reentry.[11] Not all clinical arrhythmias have been reproducible during electrophysiologic studies of tumor-associated VT. Lack of inducibility suggests that the tumor might act as a focus of enhanced automaticity.[12]

Tumors associated with ventricular arrhythmias can be benign or malignant. Published reports indicate VT's predominant association with benign primary cardiac tumors, including myxomas, hemangiomas, rhabdomyomas, lipomas, papillary fibroelastomas, fibromas, calcified amorphous tumors, and proliferative angiomatosis.[13-20] The ages of affected patients have ranged from 3 months to 78 years.[12,21] Among malignant primary tumors associated with VT are B-cell lymphomas, T-cell lymphomas, and osteosarcomas.[22-24] Although most cardiac tumors are metastatic, metastasis is much less frequently associated with VT. Perhaps patients' rapid progression to death decreases the likelihood that metastatic cardiac tumors would have time to become a source of ventricular arrhythmia. No direct correlation of tumor size to the development of VT has been made; however, most metastatic lesions associated with ventricular arrhythmia are large. We found only one report of a tumor smaller than 1.8 cm in its greatest dimension[27]—and, in most cases, the tumor was substan-

**TABLE I.** Metastatic Tumors Causing Ventricular Tachycardia

| Reference | Pt. Age (yr), Sex | Tumor Type | Tumor Size (cm) | LV Dysfunction (EF <0.50) | Tumor Location | Treatment | Outcome of VT |
|---|---|---|---|---|---|---|---|
| Sheldon R and Isaac D[9] (1991) | 35, F | Melanoma | 5 × 4 × 2 | No | Posterolateral LV | Procainamide | Suppressed |
| Leak D[25] (1998) | 66, M | Lung | N/A | Yes | RV free wall | Amiodarone | Suppressed |
| Manojkumar R, et al.[26] (2001) | 12, M | Non-Hodgkin lymphoma | 2.6 × 2.7 | N/A | RVOT | CHOP | Cured |
| Cho JG, et al.[27] (2002) | 39, F | Non-Hodgkin lymphoma | 1 × 1.7 and 2.3 × 3.1 | N/A | Septum and posterior near MV | CHOP | Cured |
| Jaster M, et al.[28] (2006) | 64, M | Leiomyosarcoma | 4 × 3 × 3 | No | Anterolateral pericardium | Chemo | Suppressed |
| Lim HE, et al.[29] (2006) | 60, M | Lung | 4 × 3 | N/A | Apical lateral LV | Amiodarone and chemo | Suppressed |
| Dorfman FK, et al.[30] (2006) | 16, M | Rhabdomyo-sarcoma | 0.4 | No | Ventricular septum | Amiodarone and ablation | Failed ablation |
| Ibars S, et al.[31] (2007) | 49, M | Squamous cell carcinoma | 2.2 | No | Posterolateral LV | Chemo | Unknown |
| Tekbas E, et al.[32] (2010) | 17, F | Osteosarcoma | 2.4 × 3.5 × 2.8 and 1.8 × 0.8 × 0.8 | N/A | LV apex | Amiodarone | Suppressed |
| Chemello D, et al.[33] (2011) | 27, F | Non-Hodgkin lymphoma | N/A | N/A | Basal septum | Chemo | Cured |
| Casella M, et al.[34] (2011) | 27, M | Gluteal sarcoma | N/A | Yes | LV anterior/lateral wall, anterior papillary muscle | Ablation | Cured |
| Lanzillo T, et al.[35] (2011) | 50, M | Renal cell | 3.6 × 2.7 × 4.8 | N/A | Anteroapical LV | ICD, chemo, and amiodarone | Suppressed |
| Huang SH, et al.[36] (2011) | 25, M | NK T-cell lymphoma | N/A | Yes | Posterolateral LV | ICD and amiodarone | Ongoing VT and ICD storm |
| Domínguez-Pérez L, et al.[37] (2012) | 64, M | Renal cell | 3.4 × 3.5 | No | Basal and mid anterior LV | Amiodarone | Cured |
| Current case | 52, F | Sarcoma | 5.6 × 2.9 | Yes | RV free wall | Amiodarone | Suppressed |

Chemo = chemotherapy; CHOP = cyclophosphamide, adriamycin, vincristine, prednisone chemotherapy; EF = ejection fraction; F = female; ICD = implantable cardioverter-defibrillator; LV = left ventricle; M = male; MV = mitral valve; N/A = not available; NK = natural killer; Pt = patient; RV = right ventricle; RVOT = right ventricular outflow tract; VT = ventricular tachycardia

tially larger (Table I).[9,25,26,28-37] Metastatic melanomas, lymphomas, rhabdomyosarcomas, leiomyosarcomas, osteosarcomas, and oral, renal, and lung cancers have been associated with VT.[9,25-33,35-37]

## Management of Tumor-Induced Ventricular Tachycardia

Ventricular tachycardia from cardiac tumors can be managed initially like VT from other origins, with use of defibrillation for unstable VT and antiarrhythmic therapy for hemodynamically stable patients. Various long-term management approaches have yielded mixed results in suppressing or eliminating recurrent VT associated with cardiac tumors.

*Conservative Therapy.* When VT is associated with rhabdomyoma, the tumor's spontaneous resolution usually results in elimination of the VT; accordingly, conservative therapy can be considered.[38] Therapy with β-blockers has been reported to suppress VT in one such instance, as well as in VT associated with other tumors.[12,39,40]

*Antiarrhythmic Therapy.* Amiodarone suppressed recurrent VT in our patient, as well as in others.[10,25,29,32] However, aggressive antiarrhythmic therapy, although often necessary, is not always successful: amiodarone and tocainide failed to prevent sudden cardiac death in a patient who had osteosarcoma.[24]

*Resection.* When feasible, resection—especially of benign primary tumors—is preferred, and it usually results in complete remission of VT.[13-18,20] Resectability depends on the tumor's location and its proximity to the coronary arteries and other vital structures. In a patient

with lipoma and recurrent VT, additional cryoablation at the resection border was performed, because prior resection had not prevented recurrent VT.[41] When malignant primary tumors do not cause death secondary to ventricular arrhythmia, they generally have poor prognoses. The median survival period of patients without metastases is 15 months. Complete excision has extended the median survival period to 17 months, compared with 6 months in incomplete resections. Accordingly, in the absence of widely metastatic disease, resection is recommended if technically feasible.[4,42] However, surgical resection is often not an option for patients with metastatic cancer.

*Chemotherapy.* Chemotherapy targeting the tumor itself has been of benefit.[28] Combined cyclophosphamide, adriamycin, vincristine, and prednisolone chemotherapy, the mainstay of treatment for non-Hodgkin lymphoma, has eliminated ventricular arrhythmias through tumor regression.[26,27,33] Nevertheless, despite adequate treatment of lymphomas, residual necrosis secondary to tumor regression can still create a nidus for continued ventricular arrhythmias.[23]

*Ablation.* Radiofrequency ablation has been used to treat unresectable tumors that cause VT, in one instance eliminating incessant VT in a 3-month-old girl who had rhabdomyoma.[21] The only reported case in an adult involved a man who had metastatic gluteal sarcoma involving the left ventricular anterolateral wall.[34] The patient was burdened by multiple episodes of drug-refractory VT and ventricular fibrillation. Pace mapping and activation mapping were used, and catheter ablation at the site of earliest activation eliminated the VT. Not all ablation has been successful, such as in a 16-year-old boy who had bidirectional VT and metastatic paratesticular rhabdomyosarcoma.[30] The authors attributed the unsuccessful result to an inability to create lesions deep enough to ablate the arrhythmia.

*Defibrillation.* Cardiac defibrillators[17,35,36] should be reserved for patients with recurrent episodes of unstable VT, a reasonable quality of life, and a life expectancy longer than one year.[43] A portable external defibrillator, such as the LifeVest, might be an option for patients who have potentially unstable arrhythmias and a poor prognosis but an otherwise reasonable quality of life.

To our knowledge, this is the first report of VT associated with radiation-induced undifferentiated sarcoma.

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