descending (LAD) artery lesion. The patient exercised to stage 5 of a Bruce protocol when significant flat ST depressions consistent with ischemia developed. A wide complex tachycardia then developed in the patient at a rate of 280 beats per minute. The patient became mildly lightheaded, but his symptoms resolved when the tachycardia resolved in less than 30 s.

The patient was referred for cardiac catheterization at that point to rule out significant coronary artery disease. Left heart catheterization revealed end-diastolic pressure of 8 mm Hg. There was no mitral valve prolapse. Left ventricular contractility was normal. The coronary arteries were normal except for systolic compression of the mid LAD (Fig 1 and 2). The patient was then referred for an electrophysiologic study. Ventricular tachycardia was induced only during isoproterenol (Isuprel) infusion. The patient was then discharged from the hospital on a regimen of β-blocker therapy and has remained asymptomatic.

**Discussion**

The congenital anomaly of a partial intramyocardial course of the LAD artery was first described by Crainiciu in 1922. Portsmann and Iwig first described systolic compression of a coronary artery on angiogram in 1960. In 1976, Noble et al described the physiologic effect of this anomaly. Severe systolic compression was shown to cause ischemia during rapid heart rates as myocardial lactate production, ST depressions, and angina were induced during atrial pacing. Shortly thereafter, there was anatomic confirmation that systolic "milking" or compression of the LAD artery is due to a partial intramyocardial course. This was described when two patients underwent coronary artery bypass surgery for severely symptomatic myocardial bridging.

To date and to our knowledge, exercise-induced ventricular tachycardia secondary to myocardial bridging has not been described. Presumably, the cause of the arrhythmia is myocardial ischemia in this case. Classic "ischemic" ST depressions appeared prior to the onset of ventricular tachycardia. Noble et al demonstrated myocardial lactate production in similar patients with myocardial bridging and ST depressions. Another potential mechanism is exercise-induced spasm in conjunction with the myocardial bridge. An ergonovine challenge was not performed in this case.

In conclusion, we have described a case of exercise-induced ventricular tachycardia in a patient with a myocardial bridge. The stress test showed evidence of ischemia with classic ST depressions prior to the onset of the arrhythmia. The electrophysiologic study also suggested an ischemic basis of the arrhythmia as the patient was inducible into ventricular tachycardia only during an isoproterenol infusion. Systolic compression of the LAD artery can be a cause of exercise-induced ventricular tachycardia.

**References**


**Metastatic Melanoma to the Heart Presenting with Ventricular Tachycardia**

Robert Sheldon, M.D., Ph.D.; and Debra Isaac, M.D.

*From the Division of Cardiology, Department of Medicine, University of Calgary, Calgary, Alberta, Canada.
†Canadian Heart Foundation Scholar. Supported by grants from the Medical Research Council of Canada, The Alberta Heart and Stroke Foundation, and the Alberta Heritage Foundation for Medical Research.
Intracardiac tumors in adults are uncommon, and the association of these with ventricular tachycardia is even more rare. We report a case of an intracardiac metastatic melanoma in a woman who presented with syncope due to ventricular tachycardia. (Chest 1991; 99:1296-98)

Intracardiac tumors may present with a variety of manifestations, including tachyarrhythmias.1 The reported tachyarrhythmias generally occur with atrial tumors, and to our knowledge, there have been no reports of sustained ventricular tachycardia associated with intracardiac tumors. Herein we report the case of a patient with an intracardiac metastatic melanoma whose manifestation was sustained ventricular tachycardia.

Case Report

A 35-year-old woman presented with one episode of unresponsiveness that lasted 15 minutes and that was followed by a period of slowed speech and decreased visual acuity. For the previous three months, she had had brief episodes of palpitations that were associated with dizziness and blurred vision. Also, she had had frequent episodes of migratory monoarthralgias that lasted two to three days, had small tender areas on her distal fingers, and small reddened areas on her hands and left foot. She had lost 6.3 kg over these three months. Ten years previously she had had a superficial spreading malignant melanoma removed with wide excision from her right paravertebral region. No recurrence had been noted.

Physical examination revealed her to be alert and oriented. She had a visual field defect in the left upper quadrant of the right eye but results of neurologic examination were otherwise unremarkable.

Her 12-lead ECG (Fig 1, top) showed right axis deviation and marked inferolateral repolarization changes. While in the coronary care unit, she had recurrent episodes of pleomorphic wide QRS complex tachycardias (Fig 1, bottom) at rates of 150 to 300 beats per minute associated with nausea, presyncope, and tinnitus. Her tachycardia was suppressed with intravenous procainamide. An echocardiogram (Fig 2) showed a large mass attached to the posterior wall of the left ventricle in the region of the posterior papillary muscle. The patient underwent surgery at which time a 5 × 4 × 2-cm mass was removed. The mass was a metastatic melanoma that had infiltrated into the myocardium in a stellate fashion. Postoperatively the patient required left ventricular assistance, but with histologic confirmation of metastatic melanoma, the mechanical assistance was discontinued and the patient died.

Discussion

Although arrhythmias have been said to be common manifestations of intracardiac tumors, we are unaware of a
previously reported association between ventricular tumors and sustained ventricular tachycardia in adults. This patient may be the first such case reported. There are two types of intracardiac tumors known to be associated with ventricular arrhythmias. Myocardial hamartomas in infants may present with incessant ventricular tachycardia that is unresponsive to medical therapy, and surgical excision is frequently curative. In adults, mesothelioma of the atrioventricular node has presented as unexplained heart block, ventricular tachycardia, or sudden death. Other intracardiac space-occupying lesions also have been associated with arrhythmias. Lipomatous hypertrophy of the interatrial septum is thought to cause various atrial arrhythmias.

The mechanism of ventricular arrhythmias associated with intracardiac space-occupying lesions is not known. In one patient with a hydatid cyst, the ventricular tachycardia was inducible with programmed electrical stimulation, which suggests that a reentrant mechanism was important. In our patient, several possible mechanisms might be entertained. First, the site of insertion of the base of the tumor might be the focus of macrore-entry. Second, the insertion of the tumor into normal myocardium might sufficiently alter normal tissue architecture to cause localized dispersion of repolarization, anisotropic conduction, and microre-entry. Third, local compression of myocardial fibers or release of humoral elements might cause an automatic ventricular tachycardia. Regardless of the mechanism of tachycardia, these cases reinforce the diagnostic approach that all patients with ventricular tachycardia should be assessed for structural heart disease. Echocardiography is currently the optimum diagnostic technique. Once the condition is diagnosed, patients with intraventricular tumors and ventricular tachycardia should be referred for surgical cure.

REFERENCES


Treatment of Superior Vena Cava Thrombosis with Recombinant Tissue Type Plasminogen Activator*

Susan Greenberg, M.D.; Robert Kosinski, M.D.; and Jeffrey Daniels, M.D., F.C.C.P.

Thrombotic occlusion of the superior vena cava is an uncommon but serious complication of chronic indwelling venous catheters. Several reports have shown thrombolytic therapy with intravenous streptokinase or urokinase to be effective in the treatment of this condition. We report a case of superior vena cava thrombosis in a 53-year-old woman receiving chemotherapy for breast carcinoma through a subcutaneously implanted venous access catheter who was successfully treated with peripheral infusion of recombinant tissue-type plasminogen activator (rtPA).

(Chest 1991; 99:1286-1301)

Oclusion of the superior vena cava (SVC) is an uncommon occurrence, but it is one that carries a high morbidity and, when acute, can lead to fatal cerebral and vocal cord edema. Numerous causes of SVC syndrome have been reported, including SVC thrombosis secondary to long-term indwelling venous catheters and transvenous pacing wires. Thrombolytic therapy with streptokinase, urokinase, or anistreplase has been shown to be effective treatment of thrombotic occlusion of the SVC. To our knowledge, the use of recombinant tissue-type plasminogen activator (rtPA) for the treatment of SVC thrombosis has not been reported previously.

CASE REPORT

A 53-year-old woman was admitted to the hospital with shortness of breath and progressive swelling of her face, neck, and arms that had begun one week prior to hospital admission. Eleven months prior to admission, she had undergone a right modified radical mastectomy with a level 2 axillary node dissection for stage III B infiltrating ductal breast carcinoma. Because of poor venous access for chemotherapy, a subcutaneous port with an attached Silastic catheter was placed via her left subclavian vein ten months prior to hospital admission. The patient received cyclophosphamide, dactinomycin, and fluorouracil via the catheter every 21 to 28 days. Because of periodic difficulty in withdrawing blood from the catheter, 10,000 U of urokinase were periodically injected to fill the catheter and port. Also, catheter patency and location were confirmed with radiocontrast injection into the Huber needle. Beginning with the fourth course of chemotherapy, the patient received 4,860 cGy in 27 treatments to the right supravaculicular region and right chest wall, followed by a boost of 1,620 cGy in nine treatments to the chest wall. One month prior to hospital admission, difficulty withdrawing blood from, but not injecting saline solution into, the port was again noted. The patient had no edema or pocket tenderness. A contrast computed tomographic (CT) scan was obtained and was without evidence of SVC thrombosis.

*From the Divisions of Hematology-Medical Oncology and Cardiology, Monmouth Medical Center, Long Branch, NJ
Reprint requests: Dr. Greenberg, 279 Third Avenue, Long Branch, NJ 07740

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Treatment of Superior Vena Cava Thrombosis (Greenberg, Kosinski, Daniels)