Metastatic Renal Cell Carcinoma*  
An Unusual Cause of Syncope

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A 77-year-old man with a history of renal cell carcinoma presented with orthostatic syncope. Investigation revealed metastatic carcinoma in the left lung extending through the pulmonary veins into the left atrium obstructing the mitral orifice. On review of the literature, this case appears to be the first to describe metastatic carcinoma to the left atrium presenting as a primary cause of syncope.

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The search for the cause of syncope is often a diagnostic challenge. Many cardiac and noncardiac conditions may lead to syncope, although frequently a definite cause cannot be found.1,4 We herein report a patient with metastatic renal cell carcinoma and syncope in whom orthostatic hypotension was the first clue to the final diagnosis.

CASE REPORT

A 77-year-old man was referred to the University Hospital, Boston, for evaluation of orthostatic syncope. He initially presented to his local physician in September 1988 complaining of postural dizziness. He described a rapid course over the preceding two months beginning with exertional dyspnea. His symptoms progressed to include lightheadedness on standing and eventually frank syncope with any sudden change from a recumbent to upright position. During that visit he experienced a witnessed syncopal episode after rising from a chair. Initially he had a barely palpable blood pressure with a heart rate of 30 beats per minute (bpm), but after two minutes he developed atrial fibrillation. He was admitted to a local hospital and was placed on a therapy with digoxin. Several further episodes of orthostatic hypotension were documented, and he was transferred to our institution for further evaluation.

His history was remarkable for renal cell carcinoma initially diagnosed in 1982. At that time he underwent left nephrectomy. He presented again in 1986 with left pulmonary metastases and he underwent a left thoracotomy with wedge resection. He was known to have residual metastatic disease and was maintained on a regimen of hormonal therapy. He was receiving no medication that could lead to orthostatic blood pressure abnormalities or rhythm disturbances.

On presentation he was in atrial fibrillation with a ventricular response of 80 bpm. Findings from his physical examination were remarkable for signs of mild congestive heart failure. He had no abnormal cardiac auscultatory findings. Of note, he had no sign of intravascular volume depletion, including poor skin turgor or dry mucous membranes. On the second hospital day he experienced another syncopal episode while getting up from bed. There were no focal neurologic deficits. At that time his pulse was threadly and slow. His systolic blood pressure was 60 mm Hg and review of telemetry recordings revealed the transient development of complete heart block with a junctional rate of 30 bpm. Within one minute this resolved into atrial fibrillation and restoration of his blood pressure to 110/70 mm Hg.

A chest roentgenogram showed a large mass in the left mid lung field. Echocardiographic examination revealed a large pedunculated mass in the left atrium with a posterior superior point of origin (Fig 1). The mass was noted to intermittently prolapse into the mitral orifice. Magnetic resonance imaging of the chest was obtained which delineated extension of the pulmonary mass into the left atrium through the pulmonary veins.

As the patient could no longer sit upright without experiencing syncopal symptoms, he was taken to the operating room for palliative tumor resection. Through a left thoracotomy and under cardiopulmonary bypass, a left pneumonectomy and partial atriectomy were performed. The left atrial defect was repaired with a pericardial patch. The operative findings confirmed a focus of metastatic renal cell carcinoma in the left lung spreading via the pulmonary veins

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Figure 1. Two-dimensional echocardiogram (apical long axis view) demonstrating tumor (T) in left atrial cavity (LA); LV = left ventricle; AO = aortic root.

Figure 2. En bloc surgical specimen showing lung (L), left atrial wall (LA), and tumor (T) extending through pulmonary vein into left atrial cavity.
into the left atrium (Fig 2). The patient tolerated the procedure and was successfully weaned from the bypass pump. Unfortunately, postoperative respiratory complications ensued and the patient died.

**DISCUSSION**

The causes of syncope can be divided into cardiovascular, noncardiovascular, and indeterminable classes. Kapoor et al, in a prospective analysis of 204 patients with syncope, identified 53 with cardiovascular causes and 54 with noncardiovascular causes. In a similar study, Silverstein et al identified 39 of 108 patients with a cardiovascular cause and 18 with a noncardiovascular cause. This distinction was believed to be important as in both studies the cardiovascular group had a statistically poorer outcome. Interestingly, of 312 patients in the combined studies, no occurrence of cardiac neoplasm was identified. When described as a cause of syncope, intracardiac neoplasms are therefore quite rare.

Primary cardiac neoplasms have an incidence of 1:4,000 in autopsy specimens. The most prevalent neoplasm is the atrial myxoma. In a 15-year review of cases from the Texas Heart Institute, 17 myxomas were diagnosed. The most common presenting symptoms were dyspnea (13), orthopnea (ten), and paroxysmal syncope (nine). Murmurs were described in 16 patients. A 50-year survey at the Johns Hopkins Hospital identified 24 myxomas. In this series the most common symptoms were dyspnea (13) and chest pain (nine), though interestingly, syncope is not described.

Secondary cardiac tumors occur 20 times more frequently, usually involving the pericardium and myocardium. The selective sparing of the endocardial surface is believed to be due to a combination of mechanical forces and intrinsic properties of the myocardium itself. Secondary intracavitary tumors are even less common. In a review of 15 intracavitary lesions, only four were secondary—metastatic breast carcinoma (one), lung carcinoma (one), and multiple myeloma (one). However, the fourth involved a paravertebral sarcoma metastatic to the lung with growth via a pulmonary vein to the left atrium. Dyspnea and hemoptyes were the presenting symptoms. In other reported cases of pulmonary tumor invading the pulmonary veins and the left atrium, the most common symptom was dyspnea.

To our knowledge, this is the first reported case of metastatic carcinoma to the left atrium presenting as a primary cause of syncope. Furthermore, in no other case has the development of postural complete heart block been described. The likely pathophysiologic explanation is that the assumption of an upright position led to prolapsing of the tumor into the mitral orifice, as demonstrated by echocardiography. Subsequently, the obstruction to left atrial outflow may have produced a rise in left atrial pressure triggering vagal reflexes leading to complete heart block. Moreover, the abrupt decrease in cardiac output may have produced ischemia in the SA and AV nodes, exacerbating conduction system abnormalities. Therefore, both the obstruction to flow as well as bradycardia resulted in an abrupt decrease in cardiac output leading to marked hypotension and syncope.

Clues to the eventual diagnosis of obstructive left atrial tumor included the initial symptoms of exertional dyspnea that rapidly progressed to postural syncope associated with bradycardia, in the absence of cardiovascular medications or signs of volume depletion. Although auscultatory findings in patients with mitral obstruction by left atrial tumor may include a loud S1, a holosystolic murmur of mitral regurgitation, a diastolic rumble from blockage at the mitral orifice, or a third heart sound termed a "tumor plop," none was present in this patient. This case well illustrates the importance of a cardiac evaluation (which includes echocardiography) among individuals with cancer known to spread to the heart or lungs, who present with orthostatic hypotension, presyncope, or syncope.

**REFERENCES**


**Haloperidol-Induced Torsades de Pointes**

*M. Kriwsky, M.D.; G.Y. Perry, M.D.; D. Tarchitsky, M.D.; Y. Gutman, M.D.; and Y. Kishon, M.D.*

A patient had torsades de pointes ventricular tachycardia related to psychotherapy with haloperidol in conventional doses. The QT interval was prolonged, and shortened after the cessation of the medication and infusion of isoproterenol. Concomitantly, torsades de pointes bursts disappeared. The observation might contribute to the understanding of the mechanism of sudden death of patients during pharmacologic psychotherapy.

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**Torsades de pointes due to therapeutic dose of haloperidol has not been described previously.**

Ventricular premature beats have been well documented during treatment with different psychiatric medications, whereas haloperidol is not considered cardiotoxic in therapeutic doses. Unexplained death in young patients treated with neuroleptic drugs has been reported. However, the mechanism of arrhythmogenesis has not been demonstrated as a possible cause to prove a definite link between cause and effect.

**CASE REPORT**

A 36-year-old man with chronic schizophrenia was admitted after a syncopal episode. One week before, treatment with oral admin-

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**Haloperidol-Induced Torsades de Pointes (Kriwsky et al)**