distal end. The damage to the guidewire was probably caused by the forceful pushing and pulling of the vessel dilator during its insertion.

**DISCUSSION**

This case demonstrates a few important points which should be emphasized when the Seldinger technique is used for central vein catheterization:

1. It is preferable to remove an existing catheter before inserting a new one. If it is decided not to do so, then the existing catheter should be withdrawn out of the path of the new catheter in order to reduce the risk of entanglement.

2. The skin incision that is made over the guidewire should be to the depth of the deep fascia, thereby eliminating the need for excessive force when inserting the vessel dilator and catheter over it. This reduces the risk of damage to the guidewire, vessel dilator, and catheter.

3. While inserting the vessel dilator or catheter over the guidewire, the latter must be held absolutely immobilized. If the guidewire is advanced together with the vessel dilator or catheter, it can be damaged, resulting in difficulties with the insertion of the dilator or catheter.

4. Guidewires with a built-in safety wire only should be used.* This is a thinner straight wire, inside the flexible coil, parallel to the thicker more rigid core wire. The safety wire is attached to both the straight and J-ends of the coil, whereas the core wire is attached only to its straight end. This design prevents uncoiling, stretching, kinking, or embolization of the guidewire during traction or manipulation. Despite the use of such a guidewire in our patient, it kinked. This was probably due to incorrect usage. However, uncoiling and fragmentation of the guidewire or catheter and subsequent embolization during their removal did not occur.

In conclusion, although the knotted guidewire and catheter were safely removed this complication could have been avoided if the aforementioned recommendations were followed.

**REFERENCES**

2. Turnbull AD, Carlon G, Makowsky M, Bains M. Multipurpose central venous access using the Cordis sheath introducer system. Crit Care Med 1979; 7:30-32

---

**Pulmonary Vein Thrombosis**

Nam H. Kim, M.D.; Carlos A. Roldan, M.D.; and Bruce K. Shively, M.D.

Pulmonary vein thrombosis is difficult to diagnose clinically and requires a combination of conventional diagnostic modalities. Transesophageal echocardiography was used in the present case to readily diagnose this entity and follow thrombus regression on anticoagulant therapy. This limited experience suggests that transesophageal echocardiography may be the initial diagnostic study of choice for pulmonary vein thrombosis.

*Ches 1993; 104:624-26*

Thrombosis of the pulmonary vein with extension into the left atrium is generally thought to be a rare complication of certain primary or secondary tumors of the lung. However, this entity may be underdiagnosed because signs and symptoms are usually absent or nonspecific. In addition, the diagnosis of pulmonary vein thrombosis is difficult and requires a combination of conventional diagnostic modalities such as ventilation-perfusion scanning, pulmonary angiography, bronchoscopy, transthoracic echocardiography, and computed tomography (CT). Accurate diagnosis is important to minimize embolization in surgically resectable tumors. In nonresectable cases, anticoagulation may be an important adjunct to antitumor therapy. We report a patient with pulmonary vein thrombosis readily diagnosed by transesophageal echocardiography. Repeat transesophageal echocardiography demonstrated thrombus regression on anticoagulant therapy.

**CASE REPORT**

A 71-year-old previously healthy male smoker was admitted with a new productive cough and progressive shortness of breath. The physical examination revealed tachypnea, decreased breath sounds, lung crackles, and wheezing in the right upper and right middle lung fields. The cardiac examination was significant for a grade 2/6 holosystolic apical murmur. No adenopathy was noted. Chest radiography showed a large right perihilar mass. The CT study demonstrated mediastinal adenopathy and a 11 × 11 × 12-cm right upper lung mass extending to the mediastinum near the superior vena cava. During diagnostic bronchoscopy, atrial fibrillation developed, and the procedure was aborted. Transthoracic echocardiography to assess the cause of atrial fibrillation showed a left atrial mass and moderate mitral regurgitation. Transesophageal echocardiography revealed a 6.5 × 2.0-cm bullet-shaped, soft-tissue-density mass originating from the right superior pulmonary vein and protruding into the left atrium. Proximally, the mass appeared to completely obstruct the right superior pulmonary vein without an identifiable point of attachment (Fig 1, left). The echocardiographic characteristics of the mass suggested a thrombus and anticoagulation therapy was started.

After 8 weeks of anticoagulation therapy, the patient developed persistent epistaxis, and anticoagulation was discontinued. At the time of discontinuation, repeat transesophageal echocardiography was performed, which showed that the mass had markedly decreased in size (Fig 1, right). A diagnosis of poorly differentiated adenocarcinoma was made by needle biopsy of the lung.

**DISCUSSION**

Pulmonary vein thrombosis is most commonly associated with bronchogenic carcinoma and also may complicate

---

*From the Veteran Affairs Medical Center, Cardiology Section, Albuquerque.*
metastatic sarcoma and chondrosarcoma. Less often, pulmonary vein thrombosis may complicate lobectomy. Multiple factors probably contribute to thrombosis in pulmonary veins associated with malignancy. These include a hypercoagulable state and mechanical compression of the veins with resultant stasis and damage to the endothelium.

The diagnosis of pulmonary vein thrombosis during life is difficult. Only 4 of the 35 tumor-related cases reported to date were diagnosed before surgery or death. As a consequence, its frequency may be greater than generally appreciated. In an autopsy series, Onuigbo concluded that this entity was underdiagnosed in previous reports. Frequently reported associated symptoms such as cough, dyspnea, and hemoptysis are nonspecific. Schiller and Madge noted that 3 of 16 patients had a systolic murmur in various locations, although the significance of this observation is unknown. The morbidity and mortality attributable to unrecognized pulmonary vein thrombosis is unknown, but systemic embolization from metastatic tumor invading a pulmonary vein has been reported. Unsuspected pulmonary vein thrombosis discovered during surgery carries a grave prognosis because of the high incidence of massive embolization. Thus, preoperative diagnosis is of vital importance, since alternative techniques of pulmonary venous clamping and cardiopulmonary bypass may minimize the risk of embolization.

The high accuracy of transesophageal echocardiography in evaluating posterior cardiac structures such as the pulmonary veins has been established. However, transesophageal echocardiography currently is not an established approach to the diagnosis of pulmonary vein thrombosis. Our experience with the present case suggests that transesophageal echocardiography may be the initial diagnostic study of choice for this entity.

Little is known regarding the treatment of pulmonary vein thrombosis. Thrombectomy has been tried successfully for thrombosis after lobectomy, but few data exist on thrombectomy for pulmonary vein thrombosis due to malignancy. Warfarin therapy for pulmonary vein thrombosis has not, to our knowledge, been previously reported. In this case, we documented its effectiveness by showing thrombus regression without clinical evidence of embolization during therapy. Clearly, further data are needed to evaluate the risks and benefits of anticoagulation therapy in pulmonary vein thrombosis, but our limited experience suggests that anticoagulation could minimize the risk of embolization in patients with nonresectable tumors. Whether anticoagulation should be offered to patients before surgery to reduce thrombus size is unknown.

REFERENCES
9. Boland TW, Winga ER, Kalfayan B. Chondrosarcoma: a case
Reversal of Alcoholic Cardiomyopathy in a Patient With Severe Coronary Artery Disease*

Venkatram Nethala, M.D.; Edward J. Brown, Jr., M.D.; Charles R. Timsol, M.D.; and Raj Patcha, M.D.

Alcohol is a known cause of cardiomyopathy. Although the mechanism is not clearly understood, abstinence prior to the onset of fibrosis has been associated with improvement in left ventricular function. As shown in this report, the presence of severe coronary artery disease should not exclude other causes of left ventricular dysfunction, especially alcoholic cardiomyopathy. (Chest 1993; 104:626)

When left ventricular dysfunction is associated with severe coronary artery disease, a causal relationship is often assumed. Although alcohol is a known cardiac toxin that can cause left ventricular dysfunction, evidence that a modest consumption of alcohol can reduce the risk of coronary heart disease may encourage the use of alcohol in cardiac patients. The following report demonstrates the effects of alcohol and abstinence from alcohol on left ventricular dysfunction.

Case Report

A 60-year-old nonsmoking business man drank 1 to 4 martinis per day for the past 10 to 15 years. He presented to his physician with symptoms and signs of congestive heart failure of 3 months' duration. Heart failure was managed with conventional therapy (diuretics, digoxin, nitrates, and captopril). Abstinence from all alcohol was started by the patient. A baseline evaluation consisted of a radionuclide ventriculographic left ventricular ejection fraction of 11 percent at rest and 11 percent after 7 minutes of supine bicycle exercise. The left ventricle was diffusely hypokinetic. Ambulatory monitoring revealed 68 ventricular premature beats per hour, 1 run of nonsustained ventricular tachycardia, and 14 ventricular couplets. Cardiac catheterization revealed a diffusely hypokinetic left ventricle with an ejection fraction of 15 percent and severe coronary artery disease (3-vessel disease with 80 percent stenosis in the proximal left anterior descending artery, 80 percent stenosis in the proximal left circumflex artery, and 80 percent stenosis in the proximal posterior descending artery). A thallium stress test was negative for infarction and ischemia.

After 8 months of abstinence from alcohol, the radionuclide left ventriculographic ejection fraction improved to 29 percent at rest and 36 percent after 12 minutes of exercise. The left ventricle was diffusely hypokinetic. After 18 months of abstinence from alcohol, the ejection fraction was 61 percent at rest and 74 percent after 12 minutes of exercise. A repeat cardiac catheterization revealed a left ventricular ejection fraction of 76 percent with no wall motion abnormalities and unchanged coronary anatomy. Repeat ambulatory monitoring detected no significant arrhythmias. The patient did not undergo coronary artery revascularization, and no changes were made in his exercise or dietary habits (other than alcohol consumption) during the 18-month period of recovery.

Discussion

The exact mechanism of cardiac dysfunction due to alcohol is not known. Cardiac toxicity may be due to the direct toxicity of alcohol and its metabolites, to the toxic effects of additives in ethanol, or to nutritional deficiencies. Alcohol and its metabolite, acetaldehyde, interfere with many cellular functions, including mitochondrial oxidation and myocyte protein and lipid metabolism. Alcohol is known to cause acute and chronic myocardial depression, even when ingested in small quantities. The prognosis of patients with alcoholic cardiomyopathy is poor with continued use of alcohol. Mortality as high as 42 percent in 36 months has been reported with continued use of alcohol. Improvement of left ventricular function after 1 year of alcohol abstinence was reported in 2 patients. 1 documented with radionuclide ventriculography and the other with cardiac catheterization and left ventriculography. Two other case reports described improvement of left ventricular function by echocardiography over a period of 10 to 14 months of abstinence from alcohol. In another patient, left ventricular function demonstrated by radionuclide ventriculography and echocardiography improved over 3 years. All of these patients had no evidence of coronary artery disease.

The short duration of symptoms before initiation of therapy and total abstinence from alcohol have been associated with a favorable outcome. Both criteria were present in our patient. The time course of improvement of left ventricular function in our patient with coronary artery disease is similar to the time course of recovery reported in patients without coronary artery disease. Alcoholic cardiomyopathy should be considered even in the presence of severe coronary artery disease and a modest amount of alcohol consumption. Improvement in left ventricular function following cessation of alcohol consumption can occur in 8 months, and complete recovery can be achieved in 18 months.

References


*From the Cardiology Division, Department of Medicine, Nassau County Medical Center, East Meadow, NY