SELECTED REPORTS

Carcinoma of the Lung Causing Pulmonary Arterial Stenosis*

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A patient is described in whom carcinoma of the lung
caused compression and obstruction of the left and right
main pulmonary arteries and auscultatory features of
peripheral pulmonary arterial stenosis.

Herein we describe findings in a man with carcinoma
of the lung causing auscultatory features of pe-
ripheral pulmonary arterial stenosis. To our knowledge,
such a patient has not been described previously.

Case Report

A 55-year-old man was healthy until January 1977 (11
months before death), when left anterior chest pain and
dyspnea first appeared. He had smoked a package of cig-
arettes daily for 30 years. The mean jugular venous pressure

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was equivalent to about 6 cm of water, and a prominent A
wave was visible. The carotid arterial pulse was normal. The
breath sounds were decreased, and the percussion note was
dull over the left chest. A right ventricular impulse was easily
palpable. The second heart sound at the base was widely
split on inspiration, and never became single during ex-
piration (Fig 1). The pulmonary component of the second
heart sound was increased in intensity. A third heart sound, $S_3$,
was present over the right sternal border. A grade 3/6 systolic ejection
murmur was heard over the precordium, both axillae and
back, loudest over the second right and left intercostal spaces,
and the murmur increased in intensity with inspiration, espe-
cially along the right sternal border and in the right axilla. No
diastolic murmurs were heard. An ECG showed prominent
left ventricular voltage and nonspecific ST segment and T
wave changes. His chest roentgenogram (Fig 2) disclosed
complete opacification of the left lung field. An echocardi-
ogram was consistent with pulmonary hypertension.

Tissue obtained from the left main stem bronchus showed
small-cell carcinoma. At right-sided cardiac catheteriza-
tion, the pressures (in mm Hg) were as follows: right atrium, $a =
8$, $v = 7$; right ventricle, $55/10$; pulmonary trunk, $55/10$;
proximal right pulmonary artery, $55/10$, and distal right
pulmonary artery, $22/10$ (Fig 1). Right atrial angiogram
(Fig 2) disclosed normal right atrial and ventricular cavities
and pulmonary trunk, but severe narrowing of the left main
and moderate narrowing of the right main pulmonary arter-
ies.

The patient received chemotherapy and local irradiation,
and a chest roentgenogram four weeks later showed consid-
erable clearing of the left lung field. Moreover, the intensity
of the systolic ejection murmur had decreased to grade 1/6.

![Figure 1. Phonocardiogram (upper) recorded at second left (2L) and right
(2R) intercostal spaces showing systolic murmurs (SM) and a persistently
split second heart sound ($S_2$). $A_2=$ aortic component of $S_2$, $CP =$ external
carotid pulse; $P_2=$ pulmonic component of $S_2$; $S_1 =$ first heart sound; $S_3 =$ third
heart sound. Pressure tracing (lower) with pullback from distal to proximal
right main pulmonary artery (RPA).]
He slowly deteriorated, however, and died ten months later (November 1977). His only evidence of right-sided congestive heart failure at any time was the presence of a third heart sound, which varied with respiration.

At necropsy, both right and left lung parenchyma and pleura were extensively replaced by carcinoma (Fig 3). The left main bronchus and pulmonary artery were severely compressed by tumor. The right main pulmonary artery was partially compressed by surrounding tumor. A large cavity filled with necrotic tumor was present in the upper left lobe. Histologic sections showed tumor invasion of the walls of the right and left main pulmonary arteries and intrapulmonary arteries. The heart weighed 420 g, and the right ventricle was dilated.

**DISCUSSION**

Although many reports have described exterior compression of one or more major extrapulmonary pulmonary arteries by neoplasm, auscultatory evidence of obstruction to one or more major pulmonary arteries has not been documented previously. In our patient, total obstruction to the left main pulmonary artery and partial obstruction to the right main pulmonary artery was documented by both angiography (during life) and

**FIGURE 3.** Drawing of heart and lungs at necropsy. Lumen of left main pulmonary artery totally occluded by carcinoma, compressing it externally, and lumen of right main pulmonary artery partially obstructed by similar mechanism. Both right ventricle and right atrium are dilated.

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necropsy, and a 33 mm Hg peak systolic pressure gradient between proximal and distal right main pulmonary artery was found at catheterization. The wide splitting of the second heart sound at the cardiac base in our patient can be attributed to the peripheral pulmonary stenosis. There was not enough right ventricular failure (only a third heart sound) in the presence of the pulmonary hypertension in our patient to account for the wide splitting of the second heart sound.

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False Aneurysm of the Ascending Aorta Presenting as an Acute Myocardial Infarction*

A Late Complication of Aortocoronary Bypass

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After coronary bypass surgery, a 40-year-old man developed disruption of the site of cannulation of the ascending aorta. The false aneurysm which ensued presented as recurrent episodes of spontaneous angina and myocardial infarction ending in coronary death 48 days after surgery. The aortic origin of all three permeable coronary bypasses were strangled by the hematoma. Awareness of this unusual potential hazard is essential, since an early suspicion would lead to proper diagnostic interventions and reoperation.

The surgical risk of aortocoronary bypass has progressively decreased from about 8 to less than 2 percent.1,2 With the improvement of techniques and a better selection of patients, the relative frequency of unexpected death tends to increase.3 We describe an unusual clinical presentation of a false aneurysm of the ascending aorta, provoking recurrent prolonged episodes of spontaneous angina, ending in coronary death 48 days after surgery.

Case Report

Ten months after an acute anteroseptal myocardial infarction, a 40-year-old man was readmitted for evaluation of angina pectoris refractory to conventional medical therapy. Selective coronary angiograms revealed severe three-vessel disease. The distal vascular bed was acceptable for coronary bypass. The left ventriculogram showed a small anterio-axial dyskinetic area.

The surgery was performed under cardiopulmonary bypass and cryopexy of the heart. The ascending aorta appeared normal macroscopically and was cannulated just proximal to the takeoff of the innominate artery. Six aortocoronary bypass grafts were completed, with three separate aortic ostia created for the saphenous vein. Each graft made a side-to-side before its end-to-side anastomosis. A thin anterio-apical aneurysm was plicated. The site of cannulation of the ascending aorta was closed with 2-O silk using two pursestring sutures, one deep and one superficial. No Teflon felt was used. Before surgery and for the following two days, the patient received our routine antibiotics, consisting of prostaphyllin (2 gm intravenously every six hours) and streptomycin (0.5 gm intramuscularly every 12 hours).

The immediate postoperative course was unremarkable, despite a new myocardial infarction of the posteroinferior wall which was noted on the electrocardiogram. The patient was discharged on the ninth postoperative day.

Two weeks after surgery, the patient was readmitted after a 30-hour episode of supraventricular tachycardia. The rate was at 210 beats per minute at the time of admission. The patient had pulmonary edema. Electrocardiography was performed. The congestive failure responded well to conventional medical therapy. The levels of cardiac enzymes did not rise. The patient returned home after ten days.

Three weeks later, the patient came to the emergency room, complaining of squeezing pain in the anterior portion of the chest similar to the pain of his previous myocardial infarction. The ECG revealed anterior subendocardial ischemia. The level of creatine phosphokinase was increased to more than twice normal. The patient became asymptomatic shortly after admission and remained so for 48 hours. He then sustained a severe pain in the anterior portion of the chest associated with

Figure 1. Evolution of ECGs. Postoperative tracing reveals presence of new infarction of posteroinferior wall. Premortem tracing shows acute anteroseptal and inferior subendocardial ischemia.