Right Apical Lesion with Respiratory Symptoms

Myung Soo Shin, M.D.*

This 55-year-old white man was admitted to the hospital with shortness of breath, dyspnea on exertion, nocturnal dyspnea and sputum production.

*Associate Professor, Department of Radiology, University Hospitals and Clinics, University of Alabama Medical Center, Birmingham, Alabama.

The patient had been in good health until six months ago. Laboratory studies were within normal range. Figure 1 was made four years previously. Figure 2 was obtained at the time of the present admission, along with the laminagram (Fig 3).
Diagnosis: Lung Cancer Arising in an Azygos Lobe

Figure 1 shows normal appearance except for an azygos lobe. Figures 2 and 3 show opacification of the azygos lobe, without collapse. The remainder of the chest is normal.

A presumptive diagnosis of azygos lobe pneumonia was made on admission and antibiotic therapy was begun and continued for a period of approximately two weeks, with neither relief of symptoms nor regression of the apical lesion.

Right thoracotomy showed a solid mass in the azygos lobe invading the azygos vein. Frozen section revealed adenocarcinoma, and the right azygos lobe was resected. The patient was subsequently discharged to receive radiation therapy on an outpatient basis.

Azygos lobes occur in about 1 percent of anatomic specimens and are visible roentgenographically in about 0.4 percent of chest roentgenograms.1

Primary bronchogenic carcinoma of an azygos lobe is obviously a rare condition, readily confused with mediastinal tumor. I could find no previous documentation of a similar case in the literature.

REFERENCE

Reprint requests: Dr. Myung Soo Shin, Acting Chief, Radiology Service, VA Hospital, Birmingham, Alabama 35233.

Ominous Mesalliance: Inhalation of Carbon Monoxide from Motor Vehicles and Cigarettes

One hundred million motor vehicles driven currently in the United States and well over 500 billion cigarettes smoked in this country annually represent a vicious combination of machine-made and self-inflicted air pollution. Most frequent victims of this nationwide health hazard are heavy smokers accustomed to driving a car or other motor vehicle as part of their daily design for living. High temperature and humidity of the atmosphere aid rapid uptake of carbon monoxide by hemoglobin. Also, hyperventilation increases carbon monoxide uptake and decreases its removal from the blood. In subjects with emphysema, absorption of carbon monoxide is reduced because not all of the tidal air is reaching the pulmonary capillaries. In urban population, normal blood contains a small amount of carbon monoxide: 0.62-1.24 percent as carboxyhemoglobin. The affinity of hemoglobin for carbon monoxide is 250 times greater than for oxygen. Relative to the cardiovascular system, it has been noted in experimental animals that exposure to toxic concentrations of carbon monoxide brings about progressive increase in the pulmonary artery pressure, increased heart rate and systolic pressure, together with decreased metabolism. Blood pressure decreases when carboxyhemoglobin values of the blood reach 10 percent. Moreover, intermittent exposure to toxic amounts of or actual poisoning with carbon monoxide may be associated with reversible inversion of T wave, elevation of R-T segment, atrioventricular dissociation and A-V block. These changes, as well as associated degenerative sequel in some of the myocardial fibers, small hemorrhages in and necrosis of the myocardium resemble closely alterations due to severe hypoxia from other causes. In humans suffering from coronary heart disease, the extraction of oxygen from the blood by the myocardium is reduced at carboxyhemoglobin levels between 5 and 12 percent. This phenomenon may be a contributory factor to the increased incidence of angina pectoris and coronary thrombosis in the individuals involved. Another adverse result of carbon monoxide is increased viscosity of the blood.

Rabbits inhaling 0.017 percent carbon monoxide in air for several weeks develop atherosclerosis and myocardial necrosis. Atheromatous changes are pronounced in cholesterol-fed animals inhaling low concentrations of carbon monoxide as compared with cholesterol-fed animals without exposure to carbon monoxide. Astrup et al (Conference on Biologic Effects of Carbon Monoxide, New York Acad Med, 1969) observed in animal experiments that carbon monoxide rendered vessel walls more permeable, with consequent increased flow and deposition of fat in vessel walls and development of atherosclerosis. Observations in humans show that low concentration of carbon monoxide may lead to inhibition of bioelectric activity of the brain. Impairment in cognitive and psychomotor areas of the brain may be associated with carboxyhemoglobin levels between 2 and 5 percent. In the lung, hypoxia caused by carbon monoxide inhibits the function of alveolar macrophages. This, in turn, weakens tissue defense against airborne bacterial infection. An apropos question may be posed: if the recurrent or sustained hypoxic state of the lung tissue might not favor development of bronchogenic carcinoma under the influence of carcinogens of cigarette smoke. One hundred million motor vehicles discharge 66 million tons of carbon monoxide annually. No wonder that in all major cities at busy intersections during hours of peak traffic the concentration of this harmful gas is much higher than the maximum allowable concentration, victimizing drivers, pedestrians and traffic policemen. Carbon monoxide in cigarette smoke is an incomplete combustion product even though the temperature at the burning zone of the cigarette is 884°C (1,565.2°F) while air is being drawn through the cigarette. Carboxyhemoglobin level of the blood is 4-6 percent in moderate smokers and up to 12 percent in heavy smokers. Its potential hazard can be estimated by adding these figures to those pertaining to motor vehicle drivers.

Andrew L. Banyai, M.D.

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