Ventilation-Perfusion Imaging in Sarcoidosis: Potential for Nonembolic Segmental Mismatch

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We report a case of a postpartum female on oral contraceptives who presented with chest pain and was initially treated for pulmonary embolism on the basis of a lobar mismatch on ventilation-perfusion imaging. Subsequent angiography revealed that the pulmonary artery was extrinsically compressed. Gallium-67-citrate imaging documented sarcoidosis with uptake in bilateral hilar nodes, both lungs and parotid and salivary glands.

Key Words: sarcoidosis; ventilation-perfusion imaging

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Perfusion defects in areas of the lungs with intact ventilation is characteristic of a pulmonary embolism (*1*). Documentation of such discordance by ventilation-perfusion (V/Q) scintigraphy is helpful in differentiating pulmonary vascular conditions such as embolism from parenchymal diseases. However, it is well recognized that V/Q mismatch is not specific for embolism and has been reported in other conditions (*2*) including a variety of hilar and parenchymal lung diseases. Previous authors (*3,4*) have cited sarcoidosis as a cause of abnormal perfusion lung scans, with angiography confirming pulmonary artery occlusion, presumably due to extrinsic pressure from lymphadenopathy. However, ventilation scanning was not included.

We recently studied a patient in whom both the clinical presentation and the V/Q scan simulated pulmonary embolism so closely that anticoagulant therapy was administered. Angiography and ^{67}Ga scintigraphy identified hilar adenopathy due to sarcoidosis as the cause of a V/Q mismatch.

CASE REPORT

A 26-yr-old black female was receiving Proventil inhalor treatment for asthma. Two weeks prior to admission, her shortness of breath worsened and was associated with right parasternal aching pain which was worse on bending over, and was accompanied by a low grade fever. She was 5 mo postpartum and was taking birth control pills. She was febrile (101.6°F) but physical examination was otherwise unremarkable except for scattered basal rales. No abnormal findings were recorded in the legs. Initial laboratory data: WBC 6.2 with 59% granulocytes; Hb. 11 g; Hct. 34%, platelets 405,000, on 2 liters of O_2, pO_2 112 mmHg; pCO_2 32 mmHg and pH 7.48. Serum chemistries were within normal limits, as was an electrocardiogram. Bilateral lower extremity venous sonography with Doppler showed no evidence of venous disease.

A chest radiograph (Fig. 1) showed bilateral enlarged hila. A ventilation (^{81m}Kr)-perfusion lung scan (Fig. 2) was performed on an emergency basis and showed a perfusion defect affecting almost the entire right upper lobe, with normal ventilation. This was regarded as a high probability scan for pulmonary embolism, and anticoagulation was initiated.

However, further work-up suggested active sarcoidosis. Gallium-67-citrate imaging (6 mCi) at 72 hr (Fig. 3), demonstrated increased uptake in the mediastinum, symmetric uptake in both hila and both lungs, as well as in the lacrimal and salivary glands bilaterally. This implied that the prominent hila on the x-ray were due to adenopathy rather than pulmonary arterial dilatation. Selective right pulmonary angiography revealed external compression of the upper lobe artery (Fig. 4), due to the enlarged hilar nodes.

Heparin was replaced by prednisone with resultant reduction in the adenopathy and resolution of the perfusion defect.

DISCUSSION

Our patient's clinical diagnosis of pulmonary embolism to explain chest pain, shortness of breath and fever seemed justified by hilar prominence on x-ray (assumed to repre-

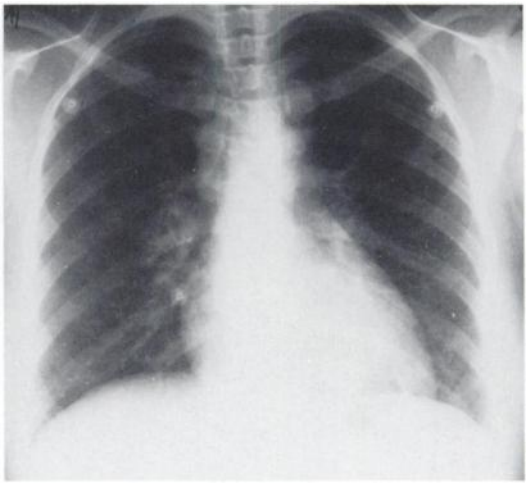

FIGURE 1. PA chest radiograph. Bilateral hilar and superior mediastinal adenopathy.

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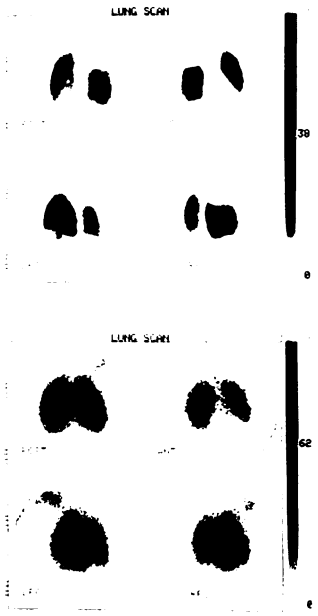

FIGURE 2. Ventilation and perfusion lung scan. Large V/Q mismatch involving almost the entire right upper lobe.

sent prominent vasculature) and by a right upper lobe mismatch on lung scan, regarded as "high probability for pulmonary embolism" by all classifications (*5*). Subsequent evaluation, including gallium imaging, indicated active sarcoidosis (*6*) and angiography showed the right pulmonary artery was extrinsically compressed due to enlarged hilar nodes.

Similar findings of reduced perfusion and pulmonary artery obstruction associated with hilar adenopathy or tumor masses have been reported in histoplasmosis (*7*) and carcinoma of the bronchus (*8*). Prior reports of pulmonary artery obstruction by sarcoid lymphadenopathy did not include ventilation scanning. Nonembolic causes of V/Q mismatch are clinically regarded as unusual, yet sarcoidosis may more commonly cause such discordance than is generally recognized. A comparison of chest radiographs and perfusion scans in 31 cases of sarcoidosis (*9*) revealed two patients who had decreased perfusion with clear lungs distal to hilar adenopathy.

Another study (*10*), investigating regional distribution of ventilation and perfusion in 11 patients with sarcoidosis, reported high V/Q areas in five patients. These patients were described as being in stage II or III, so presumably the abnormality was not associated with hilar adenopathy. Not surprisingly, regions of low V/Q were also found in 4

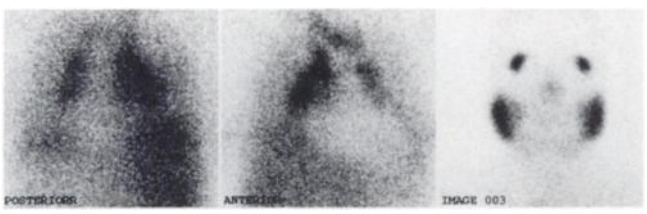

FIGURE 3. Gallium-67 radionuclide scan. Uptake in lungs, mediastinum, both hila ("lambda sign") and lacrimal and parotid glands ("panda sign").

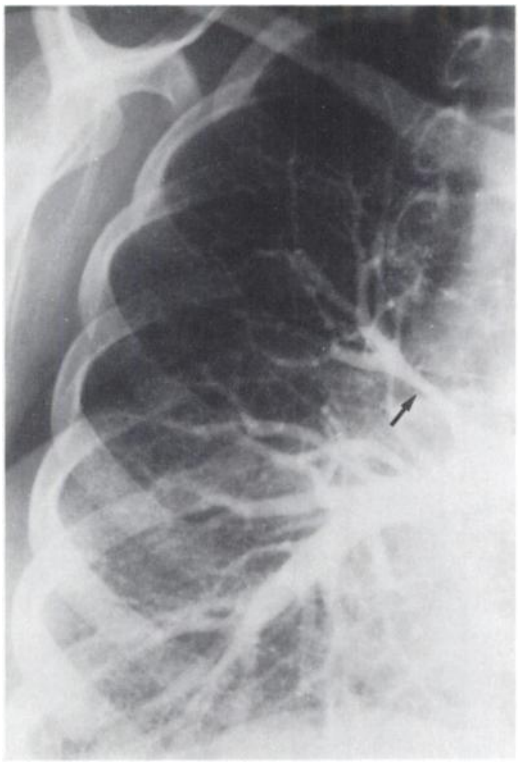

FIGURE 4. Selective right pulmonary angiogram. Extrinsic compression of the upper lobe artery.

of the 11 patients, presumably due to airway obstruction. Others (*11*) reported four patients with mismatches among 34 patients with nonembolic pulmonary diseases. Similar V/Q scan findings have been observed in idiopathic pulmonary fibrosis and attributed to fibrosis and occlusion of small blood vessels (*12*).

Although a long gamut of nonembolic mismatches have been reported, few present in distributions similar to pulmonary embolism. Embolic mismatches tend to be lobar or segmental, wedge-shaped and pleural based, reflecting defective perfusion caused by a blocked artery or branch. Hilar and mediastinal adenopathy are quite likely to produce defects identical in distribution to embolism. Hilar sarcoid lymphadenopathy may represent an unrecognized rather than uncommon cause of V/Q mismatch, which can be easily misinterpreted as pulmonary embolism.

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Condensed from
30 Years Ago

Technetium-99m-Pertechnetate for Brain Scanning

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Element No. 43, the metal technetium, "the artificial one," was discovered by Segré in 1937. This element was so named because it does not exist in nature. All of its nuclides are radioactive. Together with manganese and rhenium, it forms group VII A of the periodic table of elements, and its chemical behavior is similar to rhenium. Similarities have been noted in the biological behavior between pertechnetate ions and the halogens, which form group VII B of the periodic table. Concentration of pertechnetate in the thyroid of animals was demonstrated by Baumann. In 1963, the gamma emissions of the nuclide ^{99m}Tc were first used clinically for visualization of the liver by scintillation scanning, following administration and hepatic localization of the parent nuclide ^{99}Mo. Harper et al. first administered ^{99m}Tc parenterally for scintillation scanning of the thyroid gland and later for localization of brain tumors. Since January 1964, we have used this material routinely for brain scanning because of its ideal physical characteristics. The short physical half-life of 6 hr, the absence of beta emission and the gamma emission of 140 keV permitted the administration of large amounts of radioactivity (1 to 10 mCi) without excessive irradiation of the patient. Because of the higher counting rates obtained, the statistical variations in count rate were minimized, and the technique of brain scanning was much improved compared with older agents such as ^{131}I-serum albumin and ^{203}Hg or ^{197}Hg-chlormerodrin.

We discuss the applicability of ^{99m}Tc-pertechnetate as an agent for brain scanning, its tissue distribution in animals and man, and the relative merits of oral versus intravenous modes of administration.

In a series of 133 patients, ^{99m}Tc-pertechnetate produced significantly better brain scans technically than the older agents ^{131}I-albumin or ^{203}Hg-chlormerodrin. Although clinical experience with this agent is still limited, it would appear that certain tumors frequently missed with older agents, such as low-grade astrocytomas and supracellular cysts, also may be missed with pertechnetate. The concentrations of this material in transplantable gliomas and normal brain tissues of mice were similar in magnitude to those obtained with the older agents.

The superiority of labeled pertechnetate appeared to be due entirely to its physical characteristics, i.e., essentially monoenergetic gamma emissions of 140 keV, absence of beta emission and short physical half-life of 6 hr. These characteristics permitted the administration of relatively large doses of 10 mCi and a significant reduction in the procedure time; yet the radiation doses were kept below the levels obtained with other agents.

The radioactive preparation is easily obtained on a daily basis from a commercially available ^{99}Mo-^{99m}Tc generator. In this laboratory, the intravenous mode of administration is preferred over the oral, although both methods of administration have been tried. The intravenous method produces more consistent results, somewhat higher count rates with the same amount of radioactivity administered, and slightly lower fractions of the administered radioactivity are excreted in the feces. The oral route, however, may be preferred at other institutions when immediate sterilization of the radioactive material cannot be carried out conveniently. By the oral route, satisfactory gastrointestinal absorption can be obtained in approximately 90% of all patients. Furthermore, with oral administration, pyrogen-free reagents need not be used, and the volume of the eluate is not critical.

It is hoped that the use of this radionuclide in the near future can be further simplified by the development of an automatically timed elutor-titrator.

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