Primary Pulmonary Hypertension in HIV Infection

To the Editor:

I read with interest the article by Speich et al., which appeared in the November 1991 issue of Chest. The authors reported six cases of primary pulmonary hypertension (PPH) in patients with human immunodeficiency virus (HIV) infection and reviewed several other similar reports. There are several points of emphasis and comment that I would like to offer about the article.

First, while they mentioned several cases of PPH in patients with HIV infection who were not intravenous (IV) drug users, I am concerned that in the cases the authors presented the PPH may have been a result of IV drug abuse and specifically the IV use of amphetamines. Rouveix and colleagues have previously suggested this association. It remains unclear to me whether PPH in the cases reviewed can be attributed to HIV infection as the authors suggest, rather than to IV drug abuse or other associated diseases, such as hemophilia.

Second, the finding of even rare foreign body emboli in one of the two patients on whom a postmortem examination was performed suggests that factors related to the IV drug abuse can be implicated in that patient.

Finally, the authors conclude by suggesting that testing for HIV infection should be routine in patients being evaluated for PPH. While there may well be an association that will ultimately be borne out in the literature between PPH and HIV infection, I think that at this point testing for HIV should continue to be based on the history of high-risk behavior or potential exposure to blood of an infected individual.

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REFERENCES


To the Editor:

We appreciate the comments of Dr Kane on our article. We wish to emphasize, however, that PPH in our patients seems to be related to HIV infection and not to drug abuse.

First, we conducted a very detailed interview about various drugs used by our five patients. The homosexual patient had never used any drugs, and as stated in the text, none of the patients had ever used anorectic agents, including IV amphetamines. Moreover, the use of amphetamines is very rare in Switzerland.

Second, we do not agree that the scant foreign body emboli in small pulmonary arteries in one of our two patients on whom autopsy was performed may have been the cause of PPH. Most of the patients with tale-associated pulmonary hypertension reported in the literature showed extensive, primarily intravascular foreign body granulomas with characteristic angiothrombosis. In the series of Tomashefski and Hirsch, foreign material was present in the lungs of 48 of 70 IV drug abusers. Although angiothrombosis occurred in 13 instances, true plexiform lesions were present in only one patient, who also had micronodular liver cirrhosis. Only one additional patient with bacterial endocarditis of the aortic and mitral valves showed pulmonary vascular changes in the absence of extensive pulmonary angiothrombosis. The findings of Kringsholm and Christoffersen further militate against the hypothesis that vasocostriction or other prothrombotic reactions to IV drug abuse cause pulmonary hypertension. Lung sections from 33 drug addicts showed birefringent material in 94 percent, granulomas in 58 percent, and angiothrombosis in 18 percent. In no case, however, could plexiform lesions or right ventricular hypertrophy be documented.
Finally, we would like to underscore that all patients with PPH should be tested for HIV infection. Testing can no longer be based on a history of high-risk behavior, because the prevalence of HIV infection is steadily increasing in nontraditional risk categories, such as heterosexuals. In Switzerland, for instance, in 1991 17.3 percent of all new AIDS cases occurred in heterosexual patients.

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REFERENCES

Diagnosis of Mycobacterial Mediastinal Lymphadenopathy by Transbronchial Needle Aspiration

To the Editor:

I read with great interest the case report by Baron and Aranda,1 which appeared in the December 1991 issue of Chest.

In the past I was nearly daily faced with intrathoracic lymphadenopathies and with the problem of how to proceed in establishing the correct diagnosis as soon as possible. Therefore, we developed some procedures and special needles4 for peritracheal and perbronchial diagnostic puncture at the time of bronchoscopic examination. We used this technique by gas insufflation in pneumomediatinography,4 as well as for aspiration in the examination of intrathoracic lymph nodes (eg, in the diagnosis and staging of malignant tumors [with proved metastases in 58.7 percent of 1,366 lung cancer patients])5 and in cases without evident radiographic enlargement. The effectiveness of our procedure was based on an anatomic study of the topography of selected nondiseased intrathoracic lymph nodes in relationship to the tracheobronchial tree, with estimation of their mean size.

In cases of suspected sarcoidosis or tuberculosis, we used aspiration specimens for cytologic examination and preliminary rapid evaluation (the next day) by means of passive transfer of tuberculin hypersensitivity with the aid of intraperitoneal guinea pig injection, which is the first step in demonstrating virulent mycobacteria by animal inoculation. The third part of the specimen was cultured.

In 1987 we reported positive results of culture and/or guinea pig inoculation using intrathoracic lymph node needle aspiration in 18 cases (24 percent of 75 examined patients).1 In 1977 we published our success in establishing the presence of pathogenic Mycobacterium tuberculosis by culture and animal inoculation by needle aspiration of intrathoracic lymph nodes in three of 196 cases with the typical clinical picture of sarcoidosis.35

Therefore, the article by Baron and Aranda is not the first report of mycobacterial intrathoracic lymphadenopathy diagnosed by means of transbronchial needle aspiration in a medical journal or monograph.

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REFERENCES

To the Editor:

During the time when we were writing our article, our search of the English-language medical literature failed to reveal other cases of intrathoracic mycobacterial lymphadenopathy diagnosed by transbronchial needle aspiration (TBNA) in the manner we described. The search was not extended to the non-English-language medical literature; hence, we were not aware of Dr Šimeček's articles.

Nevertheless, we appreciate Dr Šimeček's pointing out that intrathoracic mycobacterial lymphadenopathy has been diagnosed by TBNA biopsy through a rigid bronchoscope. This further supports our recommendation that when performing TBNA (which, in the majority of cases, is now being done through the fiberoptic bronchoscope), aspirates should be obtained for acid-fast smear and culture if a diagnosis of mycobacterial disease is being entertained. Intrapertoneal guinea pig inoculation of aspirates, to our knowledge, is currently not being done routinely to diagnose tuberculosis.

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The APACHE III Prognostic System

To the Editor:

We are writing concerning the article by Knaus et al., which appeared in the December 1991 issue of Chest. In that article the authors compare the overall correct classification rates, based on 2 x 2 classification tables with 0.50 cutpoint, for our Mortality Probability Model (MPM)6 and the APACHE III system. The MPM system consists of distinct models at admission to the intensive care unit (ICU), at 24 h, and at 48 h. The authors correctly chose the 24-h MPM, rather than the admission MPM, as the basis for their comparison with APACHE III, but their figure of 79.1 percent total correct classification for the MPM is incorrect. That number appears in a table in our article in which values are given for patients in the ICU for 48 h or more. The accurate overall correct classification rate for the 24-h MPM, given in the text of the article, is 84.9

1919

CHEST / 102 / 6 / DECEMBER, 1992