Acute Histoplasmosis in a Hyperreactive Individual

To the Editor:

Drs. Kocielski and Byron describe an interesting case of acute histoplasmosis in the July 1993 issue of Chest and inferred that the patient's response indicated that he was "hyperreactive" in their words. I submit that they have presented a description of acute primary histoplasmosis in a young adult that is unique, not because of the hyperreactivity of the patient but rather because of the rarity of such perceptive observation and diagnosis of this clinical situation.

In clinical descriptions of primary histoplasmosis in the literature, albeit usually in younger persons the clinical/roentgenographic/pathologic syndrome described, very closely resembles the case report of Drs. Kocielski and Byron. In particular, the involvement of liver and spleen as noted in the case report is an almost uniform finding, which has established the validity of the concept of early hematogenous dissemination of the yeast during the primary infection. It is such foci that give rise to the splenic calcifications so characteristic a sign of previous histoplasmic infection. As a rule, the liver is less dramatically involved but it is not an appropriate conclusion, in my judgment, to use this as an indication of hyperreactivity.

Another point discussed in the case report was the perieradinitis encountered during bronchoscopy. Although the authors state, "More than 90 percent of patients undergoing bronchoscopy during the course of acute histoplasmosis have a normal bronchial tree..." it is well recognized that the hilar and mediastinal nodal involvement in primary histoplasmosis may be extensive, quite out of proportion to the size of the lung parenchymal involvement, and be associated with considerable peribronchial inflammation. In fact, this massive inflammatory response is considered the basis for the complications of primary histoplasmosis, which produce the many mechanical problems such as broncholithiasis, superior vena caval obstruction, bronchial obstruction with either atelectasis, bronchiecasis, or both as subsequent events, esophageal perforation/diverticulum production, pulmonary artery obstruction, etc, all of which have been repeatedly described in the literature of the 1950s, 60s, and 70s, summarized by Schwarz.

An important point was the failure to culture biopsy material and the dependence on direct smears of tissue biopsies as definitive microbiologic examinations. A culture is the only way in which the presence or absence of organisms such as Histoplasma capsulatum or Mycobacterium tuberculosis can be established with reasonable assurance. In the case described, the use of serologic tests, especially the follow-up complement fixation titer that rose as compared with the one done during the acute phase, secured the diagnosis, especially in the face of a negative tuberculin test.

Finally, although the history spoke against AIDS risk factors, I think that currently there is an indication to look for the presence of HIV antibodies in anyone with a clinical infection that behaves in a fashion that to our experience is unusual. I agree with the authors that this would almost certainly have been negative since the patient did, indeed, have a good granulomatous reaction and, albeit with amphotericin therapy, did recover.

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REFERENCES

Massive Atelectasis With Respiratory Arrest Due To Transtracheal Oxygen Catheter-related Mass Formation

To the Editor:

We read with interest the recent article in the November 1993 issue of Chest by de Groot et al. describing a nearly fatal tracheal obstruction resulting from the formation of a mucous plug around the tip of a transtracheal oxygen catheter. We also recently had a patient with a serious complication related to this type of therapy. Our patient was a 69-year-old white man with a history of oxygen-dependent COPD, a paralyzed left hemidiaphragm, and stable chronic lymphocytic leukemia. He had a transtracheal oxygen catheter placed in 1987 without significant problem. He recently presented to the emergency room complaining of a 1-week history of increasing cough, throat discomfort, and dyspnea. He was seen by the otolaryngologist who had placed the catheter originally.

Catheter removal was immediately followed by a severe coughing paroxysm with the patient leaning forward in the head-down position. He became apneic and required endotracheal intubation. Physical examination after intubation revealed absent breath sounds on the left side. Chest radiographs revealed total opacification of the left hemithorax with mild tracheal deviation to the left and an elevated left hemidiaphragm, despite appropriate endotracheal (ET) tube positioning. He was transferred to the critical care unit for mechanical ventilatory support including an FiO2 of 1.0 and a positive end-expiratory pressure level of 10. His oxygenation failed to improve, and repeat chest radiograph revealed only slight aeration of the left lung, with massive atelectasis apparent. Fiberoptic bronchoscopy revealed total obstruction of the left upper lobe airway with a thick dark brown plug. Lavage and suction maneuvers, and the usual foreign body removal techniques, were unsuccessful. Finally, the approximately 2-cm by 1-cm plug was dislodged from the left-side airway and into the right lower lobe airway, where suctioning with an 18F suction catheter via the ET tube was successful. Histopathologic examination revealed a mucous plug with occasional squamous epithelial cells.

Significant complications related to transtracheal oxygen administration have been reported infrequently and are usually minor. As experience with these two patients suggests, life-threatening events related to transtracheal oxygen catheters are possible.

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