Communications to the Editor

Communications for this section will be published as space and priorities permit. The comments should not exceed 350 words in length, with a maximum of five references; one figure or table can be printed. Exceptions may occur under particular circumstances. Contributions may include comments on articles published in this periodical, or they may be reports of unique educational character. Specific permission to publish should be cited in a covering letter or appended as a postscript.

Chlamydia trachomatis Pericarditis in a Patient with Lung Cancer

To the Editor:

Chlamydia trachomatis often infects genitourinary organs and the conjunctiva. Reports of pericarditis and pleuritis due to this organism are rare.1,2 To our knowledge, an instance of pericarditis at the onset has not been reported. We report a case of C trachomatis pericarditis and pleuritis associated with lung cancer.

A 62-year-old man has come to our hospital regularly for medical treatment for hypertension for the last four years. He complained of exertional and nocturnal dyspnea one day. His cardiothoracic ratio was increased from 50 to 57 percent, and pulmonary congestion was observed on the chest x-ray film at that time. Massive pericardial effusion was demonstrated by two-dimensional echocardiography. Because cardiac tamponade progressed after admission, pericardiocentesis was performed three times, and the pericardial effusion was eliminated. Bloody pericardial effusion was aspirated each time.

Round cells with intracytoplasmic inclusion bodies were observed in the aspirated fluids. The inclusion bodies were dyed with periodic acid-Schiff stain (Fig 1) and Alcian blue. The inclusion bodies had a positive response when the direct immunofluorescent method using monoclonal antibody for C trachomatis was used. In an immunofluorescent antibody assay, the IgG titer against C trachomatis was increased to 128-fold (normal value was less than eightfold). Specific IgM was not detectable. The titer of antibody against human T-cell lymphotropic virus was <16-fold (within normal limits).

Pericardial effusion was not collected after evacuation, but bilateral pleural effusion was increased and bloody pleural fluid with the same round cells was aspirated by puncture. Treatment with minocycline (200 mg/d injected intravenously for four weeks) resulted in disappearance of the round cells with inclusion bodies. Adenocarcinoma was demonstrated at the entrance of the left lower lobar bronchus with bronchoscopy and biopsy. The patient died of respiratory insufficiency due to cancer invasion.

Chlamydia trachomatis pneumonia has been reported in a patient seropositive for human immunodeficiency virus (HIV) infection3 and in immunocompromised patients.1,4 Our patient was not infected with HIV. The white cell count was 4,600, with a differential of 61 percent polymorphonuclear leukocytes, 5 percent band forms, 26 percent lymphocytes, and 8 percent monocytes. There was no superficial evidence of immunodeficiency. However, it is not completely impossible for cancer to suppress the immunoresponsiveness. It is important to recognize that C trachomatis pericarditis and pleuritis are potential complications of lung cancer.

Toshio Honda, M.D.,
Masahiro Kitade, M.D.,
Nobuo Ueda, M.D., and
Keizo Furuya, M.D.,
Ehime Prefectural Central Hospital,
Matsuyama, Japan

REFERENCES

2 Grayston JR, Mordhorst CH, Wang SP. Childhood myocarditis associated with Chlamydia trachomatis infection. JAMA 1981; 246:2823-27

Spontaneous Pneumothorax in AIDS Patients Not Receiving Prophylaxis against Pneumocystis carinii Pneumonia

To the Editor:

We found the article by Martinez et al., which appeared in the December 1988 issue of Chest, very interesting. They reported six cases of pneumothorax that occurred in six AIDS patients who did not receive prophylaxis against Pneumocystis carinii pneumonia.

Figure 1. Round cells with intracytoplasmic inclusion bodies observed in aspirated fluid (periodic acid-Schiff, original magnification ×1,300).
not have active *Pneumocystis carinii* pneumonia (PCP). All of these patients had a history of single or recurrent episodes of PCP and were receiving aerosolized pentamidine isethionate as prophylaxis against recurrence of PCP. It was suspected that the pneumothoraces were caused by coughing due to the irritative effect of pentamidine therapy on the airways superimposed on abnormal decreased lung compliance secondary to interstitial fibrosis caused by previous PCP.

From January 1, 1988, to January 1, 1991, 1,200 known human immunodeficiency virus-positive patients were admitted to our institution. Thirty-two patients had spontaneous pneumothorax either on admission or during hospitalization. Twenty-four patients (75 percent) with spontaneous pneumothorax were not receiving PCP prophylaxis, and only three patients (10 percent) were receiving aerosolized pentamidine prophylaxis. The incidence of spontaneous pneumothorax in our AIDS group admitted to the hospital was 2.7 percent.

At the Fifth International Conference on AIDS, Newsome et al. reported that pneumothorax occurred in eight (2.5 percent) of the 327 patients with prior PCP who had been receiving aerosolized pentamidine prophylaxis for three to 13 months; the majority (75 percent) had evidence of active PCP.

When we compared the incidence of pneumothorax in patients receiving aerosolized pentamidine prophylaxis (2.5 percent) with that in our group, who for the most part received no prophylaxis, we found no statistical difference. Although we cannot exclude the possibility that inhaled pentamidine can directly cause pneumothorax, the evidence presented more likely implicates predisposing damage from prior episodes of PCP or, more likely, ongoing tissue destruction from recurrent or active infection. It would be helpful to know whether other institutions have any significant differences in the incidence of pneumothorax in patients treated with prophylactic aerosolized pentamidine and in those who receive no prophylaxis.

Kaveh Bagheri, M.D.,
Terrance J. Truitt, M.D., and
Benjamin H. Safirstein, M.D., F.C.C.P.,
Pulmonary Division,
Saint Michael's Medical Center,
Newark, New Jersey

REFERENCES

2. Newsome CS, Ward DJ, Pierce PF. Spontaneous pneumothorax in AIDS patients on prophylactic aerosolized pentamidine. Presented at the Fifth International Conference on AIDS, Montreal, June 5, 1989

Catheter-Related Infections and Associated Septicemia

To the Editor:

We are writing to express our concern about the article by Norwood et al., which appeared in the April 1991 issue of Chest. While we agree with the authors that much of the literature addressing this topic is plagued by varying definitions of catheter-related infection and anecdotal evidence, the recommendations that arterial catheters, pulmonary artery catheters, and central venous catheters be changed only when there is evidence of catheter-related local infection or bacteremia is disturbing for several reasons. First, the data presented by the authors are derived mainly from surgical (trauma) ICU populations, which represent a younger population without prior multiple medical conditions. Extrapolation of their data to other surgical and medical ICU patients may not be valid.

Second, the work of Norwood et al. suggests that patients with more severe underlying illness (eg, sepsis) may be at greater risk for catheter-related infection; however, their recommendations do not appear to take this into consideration.

Third, catheter-related infections, including cellulitis, septic thrombophlebitis, and bacteremia, are a major cause of nosocomial infection, the risk of which increases with duration of catheterization. Catheter-associated bacteremia is associated not only with endocarditis, metastatic infection, and septic shock, but also with prolonged hospitalization and extended intravenous antibiotic therapy. The mortality from staphylococcal bacteremia varies from 21 percent to 62 percent. Prevention of these complications should be at the heart of policies regarding intravascular catheters. The incidence of catheter-related infection in our medical ICU has decreased by at least 50 percent since the institution of strict guidelines regarding catheter insertion and duration of catheterization. Only one catheter-associated infection has been reported during the past four reporting months. Furthermore, the complication rate with more frequent catheter placement has remained unchanged at less than 1 percent.

The authors' recommendation to remove central venous and pulmonary artery catheters only after a catheter-related infection is clinically evident leaves us in the less-than-desirable situation of treating, rather than preventing, potentially fatal iatrogenic complications. The only way to minimize catheter-related infection is to limit the time in which central venous and pulmonary artery catheters are in situ. We applaud the efforts by Dr Norwood and his colleagues to help clarify a controversial topic; however, we question their conclusions and the decision by the ACCP Critical Care Council to publish this paper in what appears to be the form of a policy statement.

Randolph J Lipchik, M.D., F.C.C.P., and Ralph M. Schapira, M.D.,
Pulmonary and Critical Care Medicine,
Medical College of Wisconsin,
Milwaukee

REFERENCES


To the Editor:

I thank Dr Lipchik and Dr Schapira for their comments about our review article, and I would like to reassure them that we share their concerns about catheter-related infections and associated bacteremia. Our recommendations should not be interpreted as representing a lackadaisical or hopeless attitude toward preventing infection. It is, rather, an attempt to arrive at some conclusions