anastomoses make most coronary sinus lesions clinically silent, but may explain the association of pulmonary emboli with thoracic pain, circulatory collapse, and death.2

Cardiac tamponade is a rare complication of central venous catheterization, but it has a high mortality rate. The most common mechanism is perforation of the intrapericardial portion of the superior vena cava, of the right atrium, or, less often, of the right ventricle.1 In some cases, the cause of the tamponade is unknown; however, there are several factors that can alter the pericardium or its permeability and cause fluid accumulation, such as administration of hypertonic solutions and high-pressure infusions and cardiac venous return blockade.1

It is very likely that in our patient the cardiac tamponade was due to the venous stasis caused by massive thrombosis of the coronary sinus and the right atrium. This combination of complications is very rare. Indeed, we found only one report in the literature; that case occurred in a newborn with a venous malformation.3 A control chest x-ray film obtained after the catheter placement in our case showed retrospectively the tip was in the wrong position (right atrium). Because the quality of the radiograph was poor, it was interpreted as normal. A better radiograph or the use of contrast medium would have indicated the advisability of withdrawing the catheter, and the outcome would have been good.

Most complications due to catheterization happen during the initial or repeated attempts to insert the catheter, but they can be discovered several hours, days, or even weeks after the catheter has been withdrawn, depending on the type of lesion.1,3,4 In our case, the patient experienced acute chest pain and shock due to cardiac tamponade 12 h after the catheter was removed.

Central venous catheterization is a common procedure with minimal risk if performed by skilled staff. However, it can cause severe and even fatal complications, as in this case. Some simple measures may reduce the morbidity of this technique, for example, radiologic control to make sure that the catheter tip is located 2 cm below a line joining the inferior borders of the clavicles.1

REFERENCES
2 Ducatman BS, McMahan JC, Edwards WD. Catheter-induced lesions of the right side of the heart. JAMA 1985; 253:791-95
5 Sheep RE, Guiney WB. Fatal cardiac tamponade: occurrence with other complications after left internal jugular vein catheterization. JAMA 1982; 248:1632-635

Postoperative Respiratory Failure Secondary to Pneumocystis carinii Pneumonia*

Mark S. Klepper, M.D.; Kalpalatha K. Guntupalli, M.D., F.C.C.P.; Ben Interriano, M.D., F.C.C.P.; Mark Dowell, M.D.; and Stephen B. Greenberg, M.D.

Pneumocystis carinii pneumonia (PCP) occurs frequently in individuals infected with the HIV virus. Malignancy, immunosuppressive drugs, and congenital immune deficiency may be associated with PCP. We describe a patient with stage I testicular carcinoma who developed hypoxic respiratory failure two days after retroperitoneal lymph node dissection. Pneumocystis carinii organisms were demonstrated by catheter lavage samples and confirmed on bronchoalveolar lavage. Testing for HIV antibody by ELISA and the Western blot test was negative; HIV viral culture and polymerase chain reaction were also negative. Pneumocystis carinii pneumonia is unusual in localized surgically cured malignancies without obvious immunodeficiency and, to our knowledge, has not been described as a cause of postoperative respiratory failure.

(Chest 1992; 101:1155-57)

Pneumocystis carinii pneumonia (PCP), once a rarely described entity, is now frequently encountered. Earlier case reports have referred to P carinii in previously healthy patients without coexisting disease or predisposing conditions.1-3 Presently, although HIV infection is the most common predisposing factor, prematurity, congenital immune deficiency, immunosuppressive or cytotoxic drug therapy, and malignancy (lymphoreticular malignancies more commonly that solid tumors4-6) are also reported to predispose to PCP infection. The cases of PCP occurring with malignant solid tumor are associated with prior irradiation, cytotoxic chemotherapy, or steroid therapy. This case report documents severe PCP with respiratory failure in an uncommon setting. Pneumocystis carinii pneumonia frequently causes respiratory insufficiency but, to our knowledge, has not been described as a cause of respiratory failure in a previously healthy individual in the immediate postoperative period.

CASE REPORT

A 36-year-old white man underwent left radical orchiectomy on Nov 8, 1989 for a testicular mass which was found to be a mixed seminoma, embryonal carcinoma, and yolk sac tumor. A preoperative chest x-ray film and chest, abdominal, and pelvic CT scans were normal.

On Nov 29, an elective radical retroperitoneal lymphadenectomy and incision and drainage of a left scrotal abscess were performed. The patient tolerated the procedure well and was extubated within 24 h. Arterial blood gas levels were pH of 7.36, PaCO2 of 46 mm Hg, and PaO2 of 98 mm Hg with an FIO2 of 0.4 by face mask. Forty-eight hours later, the patient's respiratory rate 45/min, and the PaO2 was 46 mm Hg with an FIO2 of 0.4 by face mask. Blood gas levels following intubation were pH of 7.32, PaCO2 of 52 mm Hg, and PaO2 of 117 mm Hg with an FIO2 of 1.0, and PEEP of 5 cm H2O. Vital signs were a temperature of 37.2°C (99.0°F), heart rate of 126

*From the Department of Internal Medicine, Baylor College of Medicine, and Ben Taub General Hospital, Houston.
beats per min, and blood pressure of 130/90 mm Hg. On physical examination, no adenopathy or thrush was noted. Examination of the lungs revealed scattered wheezes and crackles bilaterally. Cardiac rhythm was regular, without murmur or gallop. A healing midline abdominal surgical incision showed no signs of active infection. A chest x-ray film 48 h after surgery revealed bilateral alveolar infiltrates. A pulmonary artery catheter showed an initial pulmonary artery pressure of 29/14 mm Hg, with a pulmonary capillary wedge pressure of 10 mm Hg. Cardiac output was 10 liters/min, and SVR was 470 dynes-sec-cm⁻². A presumptive diagnosis of sepsis with noncardiogenic pulmonary edema was made, and broad-spectrum antibiotics were started. Cultures of blood, urine, and sputum obtained at this time were negative. On Dec 5, a wedged catheter lavage was performed using a protected suction catheter system. Approximately 60 ml of physiologic saline was used to lavage the right lower lobe, and P. carinii organisms were detected by Calcofluor white and immunofluorescent antibody techniques. Therapy with the trimethoprim-sulfamethoxazole mixture was started intravenously. Bronchoalveolar lavage of the lingula was performed four days later (Dec 9) using the fiberoptic bronchoscope, reconfirming P. carinii infection. Methylprednisolone was given at a dose of 70 mg every 6 h for a total of five days. Despite left-sided pneumothorax, the patient was gradually weaned from the ventilator and discharged after 75 days in the hospital.

Repeated ELISA and Western blot tests were negative for HIV antibody, as was HIV viral culture. A polymerase chain reaction performed 40 days after surgery was negative (performed by Dr. F. Blaine Hollinger).

**DISCUSSION**

The underlying reasons why this patient developed PCP are unclear. Suspicion of HIV viral infection was high, given the association between HIV infection and seminoma. Therefore, despite negative HIV antibody and viral culture studies, T-lymphocyte counts with subset determinations were obtained. The CD⁺/CD₈⁺ counts obtained three days after steroid therapy was started were both depressed (140/160/160 cu mm, respectively). In a recent study of patients with AIDS, CD⁺⁺ counts (200 cu mm to 250 cu mm) were shown to be strong predictors of opportunistic pulmonary infection. It is not known whether this patient's low CD⁺⁺ count reflects overall immune suppressions, postoperative changes, or effects of corticosteroids.

Many investigators have shown significant depression of lymphocyte function and T-lymphocyte subset counts following surgery. Riboli et al have shown abnormalities of cell-mediated immunity manifested as reversible anergy following surgical procedures. The immune suppression was more severe and prolonged in patients with neoplastic disease, with recovery to baseline between seven and ten days after operation. A study by Platt and colleagues, looking at the effects of surgical stress on children, showed mean CD⁺ counts of 2,600 cu mm, decreasing to a minimum of 1,150 cu mm at 12 h after surgery and 1,210 cu mm at 48 h.

The first-line therapy for PCP is the trimethoprim-sulfamethoxazole combination which was started at the time of diagnosis of PCP. Due to the lack of improvement after nine days, this was changed to pentamidine isethionate. Several studies have shown improved survival rates by using corticosteroids in patients with AIDS-related PCP. This patient received five days of methylprednisolone (280 mg daily), with a significant improvement in his gas exchange and clinical status.

In summary, this is a case of hypoxemic respiratory failure secondary to PCP occurring in the early postoperative period in a previously asymptomatic HIV-negative patient. Both the patient and clinical setting were atypical, and had the diagnosis not been pursued aggressively, specific therapy for PCP may not have been given. This suggests that PCP should be considered in the setting of hypoxemia, bilateral infiltrates, and fever, regardless of the HIV status or underlying disease, especially in the postoperative setting.

**ACKNOWLEDGMENT:** We thank Ms. Mary Holly for secretarial assistance.

**REFERENCES**

7 Hamlin WB. Pneumocystis carinii. JAMA 1968; 204:173-74
9 Price RA, Hughes WT. Histopathology of Pneumocystis carinii infestation and infection in malignant disease in childhood. Human Pathol 1974; 5:737-52
11 Slade MS, Simmons RL, Yunis E, Greenberg LJ. Immunodepression after major surgery in normal patients. Surgery 1975; 78:363-72

Pulmonary Developmental Anomaly Associated with Klippel-Feil Syndrome and Anomalous Atrioventricular Conduction*

Rajesh Bhagat, M.D.; Kaushal Runt, M.D.; Vijay K. Singh, M.B., B.S., F.C.C.P; Chandrashekar Runt, M.D.; Ajay Gupta, M.D.; and Om Prakash Jaggi, M.D.

We report the findings in a patient with Klippel-Feil syndrome and associated agenesis of right upper and middle lobes, hypoplasia of the right lower lobe of the lung, and Lown-Ganong-Levine syndrome. To our knowledge, such an association has not been previously described.

(Chest 1992; 101:1157-58)

Pulmonary agenesis and hypoplasia may be one of many multisystem anomalies in primary embryologic defect syndrome or may result from decreased intrathoracic space due to other congenital thoracic or spinal defects. Klippel-Feil syndrome, resulting from congenitally fused cervical vertebrae, has been cited to be rarely associated with pulmonary agenesis and hypoplasia. Although many congenital cardiac anomalies have been identified with all of these conditions, to our knowledge, an accessory atrioven-

*From the Clinical Research Centre (Drs. Bhagat, Punt, and Jaggi) and DST, CVM, ABC Foundation (Dr. Singh), Vallabhbhai Patel Chest Institute, University of Delhi, and the Institute of Nuclear Medicine and Allied Sciences (Drs. Punt and Gupta), Delhi, India.

![Figure 1. Bronchogram of right lung showing opaque right hemothorax with herniation of contralateral lung and absence of right upper and middle lobes and hypoplastic right lower lobe.](http://journal.publications.chestnet.org/pdfaccess.ashx?url=/data/journals/chest/21642/)

**CASE REPORT**

A 23-year-old woman was referred to us for investigation of an abnormal chest roentgenogram, prompted three months ago by a bout of respiratory tract infection. When she presented to us, she had no complaint. The patient had never smoked and never suffered from any chronic pleuropulmonary disease. Her menstrual cycle was normal and regular. She had a low hairline and a short neck with painless restriction of movement, more so on the left side. Examination of the chest revealed signs of volume loss of the right side with ipsilateral shift of the mediastinum. Heart sounds were best heard in the right parasternal region; no added sounds were audible.

The findings on routine investigations of blood and urine were within normal limits. The chest roentgenogram revealed an opaque right hemothorax with ipsilateral shift of the mediastinum and herniation of the contralateral lung (Fig 1). Roentgenograms of the cervical and thoracic spine showed block C6-7 and D3-4 vertebrae with a D2 hemivertebral. Smears and cultures of sputum for pyogenic organisms and Mycobacterium tuberculosis were repeatedly negative. On fiberoptic bronchoscopy the right main bronchus was narrow and tortuous. The right upper lobe bronchus was absent, and the right intermediate bronchus narrowed progressively, preventing negotiation beyond 4 cm. Bronchography (Fig 1) and CT of the thorax confirmed the absence of right upper and middle lobes, with a hypoplastic right lower lobe. Digital subtraction angiography demonstrated a tiny hypoplastic right pulmonary artery (Fig 2). Findings on CT and ultrasonicographic examination of the abdomen were essentially normal, and a skeletal survey did not reveal any bony abnormality.