Pleural Pseudotumoral Mass Revealing an Extrapulmonary Pneumocystis carinii Infection

Pneumocystis carinii pneumonia (PCP) is the most common opportunistic infection in patients with AIDS, affecting 60% to 80% [1]. Extrapulmonary P. carinii infection is rare (2.5% of patients) and may occur in HIV-infected patients without evidence of PCP [1, 2]. The liver, spleen, bone marrow, lymph nodes, adrenal glands, thyroid, and ear appear to be the sites mainly involved, according to postmortem studies [2, 3]. Pleural involvement is rare and has been reported in cases clinically presenting as pneumothorax or pleurisy [4–6]. We report a case of a pleural pseudotumoral mass revealing P. carinii infection.

A 30-year-old man with a history of iv drug abuse and tricuspid endocarditis was found to be HIV-positive in 1989. In 1993 esophageal candidiasis and digestive cryptosporidiosis were diagnosed, and since that time he had received didanosine (200 mg daily), spiramycin, and aerosolized pentamidine (300 mg monthly) as primary prophylaxis against PCP. He presented in January 1995 because of anorexia, a 3-kg weight loss, low-grade fever, night sweats, cough, and laterothoracic pain.

On physical examination, the right basal vesicular murmur was decreased. Laboratory examination showed the following values: hemoglobin, 140 g/L; platelets, 66,000/mm³; leukocytes, 2,000/mm³ (neutrophils, 70%; lymphocytes, 19%; CD4 cells, 0/mm³; and CD8 cells, 230/mm³); p24 antigen, 300 pg/mL; lactate dehydrogenase (LDH), 463 IU/L; alanine aminotransferase, 69 IU/L; aspartate aminotransferase, 57 IU/L; fibrin, 3.6 g/L; pH, 7.4; partial pressure of CO₂, 4 kPa; partial pressure of O₂, 13 kPa; HCO₃, 18; and O₂ saturation in arterial blood, 98%. Chest roentgenographic findings were considered normal. CT scanning revealed a heterogeneous mass in the right pleural base (figure 1), with normal parenchyma and mediastinal adenopathies. A hypodense micronodular formation was observed in the spleen.

Culture of bronchoalveolar lavage fluid did not yield any microorganisms. Blood cultures were negative, as were tests for Cryptococcus, Toxoplasma, Aspergillus, Legionella, Mycoplasma, and Leishmania species. Cytomegalovirus was present only in urine. A transparietal needle biopsy of the pleural mass was performed, and examination of biopsy specimens revealed partial necrosis and numerous P. carinii organisms. The patient received iv trimethoprim-sulfamethoxazole, which produced a good initial response; however, 3 weeks later his condition worsened, and he died. Autopsy was not permitted.

In large series of extrapulmonary P. carinii infections, pleural involvement has rarely been reported. Among 161 autopsyed patients with AIDS, extrapulmonary P. carinii infection was detected in 2.5%, but no pleural localization was found [2]. In a review of 37 patients, spontaneous pneumothorax was reported in four cases; this was possibly secondary to pleural localization of P. carinii, although no direct involvement was demonstrated [3]. Finally, in a series of 34 patients, direct pleural involvement concomitant with pulmonary vasculitis was shown in only one patient [1]. Pleural P. carinii infection is usually suspected in cases of pneumothorax or pleurisy [4–6].

A case of paraspinous mass due to P. carinii has been reported previously [1], but to our knowledge this is the first case of pseudotumoral presentation. In contrast to the rather unusual radiologic findings, the clinical presentation, moderately elevated LDH levels, severe immunosuppression, and poor evolution are common (although nonspecific) features of extrapulmonary P. carinii infection [1, 3].

The mechanisms of dissemination of P. carinii to the pleura are not clearly established, but in almost 50% of cases, this localization occurs following administration of an aerosolized prophylactic agent; therefore, poor diffusion of pentamidine to the pleura is highly suspected [2, 3]. Pneumothorax and pleural effusion usually complicate parenchymal necrosis, cavitation, and direct fistulization [7], which explains why P. carinii may be detected in pleural fluid in some cases [8].

In this patient, no evidence of PCP could be demonstrated, but isolation of P. carinii from the bronchoalveolar lavage fluid might have been difficult because of the pentamidine prophylaxis. Subpleuritic “smoldering” PCP might have been present [6]. Necrosis was present, however, as shown by the tomodensitometric and histologic findings and the increase in LDH level. In addition, a hematogenous and lymphatic diffusion of P. carinii might have occurred, which would account for the splenic lesions and mediastinal adenopathies (which were also likely due to P. carinii) [3, 4]. In conclusion, a pseudotumoral mass as

![Figure 1. Thoracic CT scan of a 30-year-old HIV-infected man who was found to have P. carinii infection; note the heterogeneous mass in the right pleural base (arrowhead) (L = liver).](https://academic.oup.com/cid/article-abstract/23/1/199/592542)
well as pneumothorax or pleurisy may reveal \textit{P. carinii} pleural infection.

G. Kaplanski, B. Granel, D. Di Stefano, J. M. Durand, and J. Soubeyrand

Service de Medicine Interne, Hopital Sainte-Marguerite et Service de Radiologie, Institut Paoli-Calmettes, Marseille, France

References


Myopericarditis Due to Parvovirus B19 in an Adult

We read with interest the article by García-Tapia et al. on parvovirus infection [1]. A large number of serologically confirmed parvovirus infections in adults have occurred in our community over the past 3 years. Most patients had a self-limiting syndrome of fever, polyarthralgia, and rashes in the absence of visceral organ or bone marrow complications. We report a case of myopericarditis due to parvovirus B19 that was confirmed by the presence of viral DNA in serum.

A 43-year-old female nurse who worked on a pediatric ward developed flu-like symptoms associated with severe diffuse joint pain without a rash in March 1994. Laboratory evaluations were negative for IgM antibodies to Epstein-Barr virus (EBV) and were positive for IgG antibodies to EBV. Her symptoms abated, but she continued to have significant polyarthralgia and low-grade fevers.

She developed severe pleuritic chest pain and supraventricular arrhythmia in October 1994. An initial two-dimensional echocardiogram showed normal ventricular contractility and a small pericardial effusion. Pericarditis was diagnosed, and the patient was treated with antiarrhythmics and nonsteroidal anti-inflammatory drugs. Her chest pain abated transiently but relapsed even though she received a short, tapering course of glucocorticoid therapy. A repeated echocardiogram 3 weeks later showed diffuse hypokinesis with an estimated ejection fraction of 45%.

Tests of acute and convalescent sera for antibodies to \textit{Toxoplasmata}, \textit{Borrelia burgdorferi}, \textit{Mycoplasma}, \textit{Chlamydia pneumoniae}, \textit{Coxiella burnetii}, cytomegalovirus, and enteroviruses showed no evidence of acute infection. A test for antinuclear antibodies was negative. Titers of IgG antibodies to parvovirus B19 were high, and those for IgM antibodies to parvovirus B19 were negative. Viral cultures of stool and throat washings were negative.

An endomyocardial biopsy was performed 6 weeks after the onset of the patient’s chest pain. Light microscopic examination of a biopsy specimen did not show any significant inflammation or viral inclusions; electron microscopy revealed many myocytes with loss of myofibrils and mitochondria (figure 1). Viral culture of the biopsy specimen was negative. Results of tests for total quantitative immunoglobulin levels, a T cell panel, and skin reactivity to mumps and tetanus antigens were normal.

Parvovirus B19 DNA was detected at high levels 3 months following the onset of chest pain even though the patient’s ventricular function had normalized. Intravenous immunoglobulin was administered daily (0.4 mg/kg for four doses), but she developed drug-related aseptic meningitis, which precluded further treatment. Repeated tests for parvovirus B19 DNA were negative on two separate occasions 2 and 4 months later. PCR analysis (performed at Specialty Laboratories, Santa Monica, CA) of a paraffin-embedded myocardial biopsy specimen was negative for parvovirus B19 DNA. There was major improvement in the patient’s condition, of a biopsy specimen did not show any significant inflammation or viral inclusions; electron microscopy revealed many myocytes with loss of myofibrils and mitochondria (figure 1). Viral culture of the biopsy specimen was negative. Results of tests for total quantitative immunoglobulin levels, a T cell panel, and skin reactivity to mumps and tetanus antigens were normal.

Parvovirus B19 DNA was detected at high levels 3 months following the onset of chest pain even though the patient’s ventricular function had normalized. Intravenous immunoglobulin was administered daily (0.4 mg/kg for four doses), but she developed drug-related aseptic meningitis, which precluded further treatment. Repeated tests for parvovirus B19 DNA were negative on two separate occasions 2 and 4 months later. PCR analysis (performed at Specialty Laboratories, Santa Monica, CA) of a paraffin-embedded myocardial biopsy specimen was negative for parvovirus B19 DNA. There was major improvement in the patient’s condition.


Figure 1. Electron micrograph of an endomyocardial biopsy specimen from a patient with myopericarditis due to parvovirus B19; significant loss of mitochondria and myofibrils is apparent in the center of the field (original magnification, $\times8,000$).