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Images in Clinical Medicine

Pneumocystis jirovecii pneumonia manifesting as a lung abscess in a woman with systemic lupus erythematosus

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Pneumocystis jirovecii pneumonia (PCP) is a potentially life-threatening infection encountered in immunocompromised individuals [1]. The radiographic appearance of a cavity in *Pneumocystis jirovecii* pneumonia is unusual [2]. Delayed diagnosis and treatment could result in a poor outcome [1]. We report a patient with systemic lupus erythematosus (SLE) who developed a cavitating pneumonia in the presence of PCP.

A 57-year-old woman with SLE, hypertension and chronic renal failure had chest pain and dyspnea for 3 days. Her medications for SLE included oral azathioprine 50 mg four times per day, oral methylprednisolone 12 mg twice daily and oral hydroxychloroquine 200 mg twice daily. In addition, she underwent two courses of pulse therapy with methylprednisolone for active lupus nephritis in the past 3 months. Her initial chest radiograph showed dense right upper lobe consolidation (Fig. 1A). The initial blood examination revealed white blood cells 3600/μL (N bands 7%, N segments 88%); creatinine 6.18 mg/dL; and C-reactive protein 35.32 mg/dL. Empirical intravenous cefepime 1000 mg every 12 hours was started, but right upper lobe cavitation ensued. The patient developed respiratory distress despite broad-spectrum antimicrobial agents. Chest computed tomography on Day 24 demonstrated a large right upper lobe cavity and diffuse mosaic ground-glass opacities (Fig. 1B). A video-assisted thoracoscopic wedge biopsy revealed consolidation of the lung. The alveolar spaces were filled with frothy alveolar exudates (Fig. 1C). Periodic acid-Schiff and Grocott's methenamine silver stains revealed *Pneumocystis jirovecii* in the alveolar spaces (Fig. 1D). Intravenous sulfamethoxzole-trimethoprim (240 mg trimethoprim every 6 hours) and intravenous methylprednisolone 40 mg every 12 hours were administered. The patient died on the 40th day of hospitalization.

The incidence of PCP among patients with acquired immunodeficiency syndrome (AIDS) has declined since the introduction of highly active antiretroviral therapy. However, PCP has increased markedly among patients with connective tissue diseases because of intense immunosuppressive treatment. The outcome has improved for AIDS patients with PCP because of a high index of suspicion and timely application of appropriate antibiotics and corticosteroids [3]. However, pulmonary manifestations in patients with connective tissue diseases are complex and the mortality among non-AIDS patients with PCP remains high, partly because of delay in diagnosis. Risk factors for patients with SLE to develop PCP are high disease activity, renal involvement, high daily corticosteroid dosage and lymphocytopenia [4]. The typical radiographic features of PCP in non-AIDS patients are diffuse, bilateral, interstitial infiltrates [5]. The initial radiographic appearance of a cavity in PCP is unusual. Lung cavitation manifests rarely in only 3.8% of patients with PCP and has been reported to simulate tuberculosis [2]. Milligan et al. described eight patients with PCP who were initially diagnosed with pulmonary tuberculosis and were started on antituberculosis treatment [6]. The differential diagnosis for pulmonary cavitary mass lesions with associated consolidation includes pulmonary tuberculosis, bacterial lung abscess, invasive fungal infection and Wegener's granulomatosis [7]. For immunocompromised hosts with nodular

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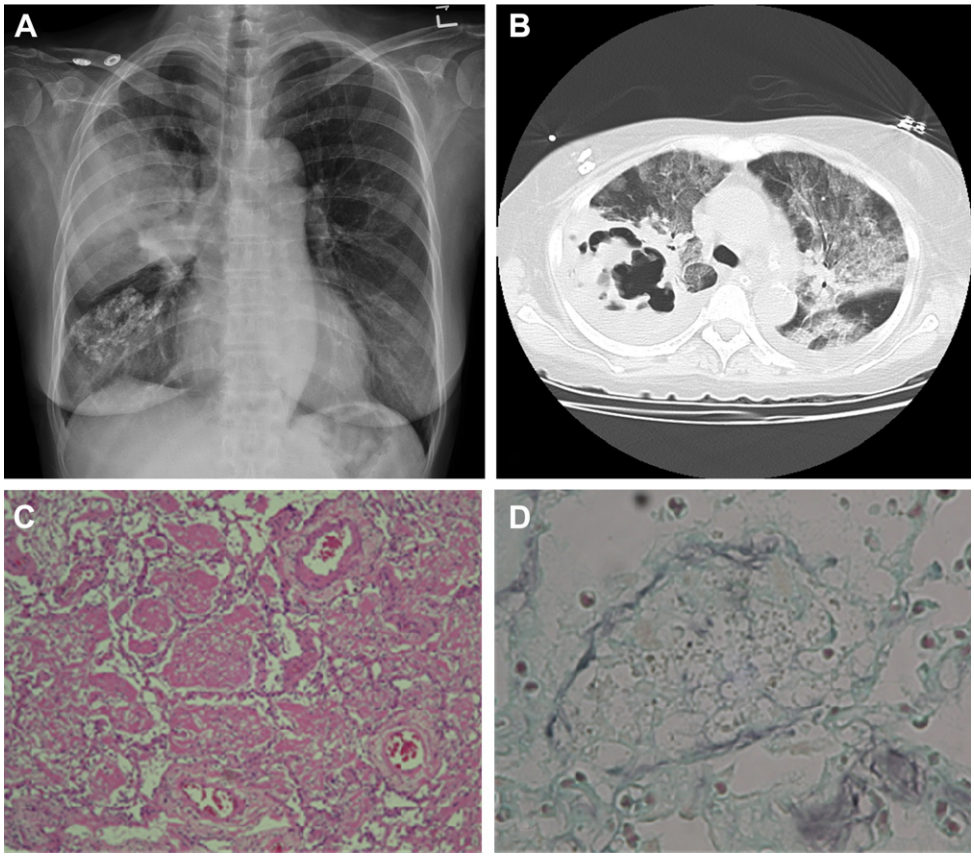

Fig. 1. (A) Initial chest radiograph shows dense right upper lobe consolidation. (B) Chest computed tomography shows a large right upper lobe cavity with diffuse mosaic ground-glass opacities. (C) Hemotoxylin and eosin stain reveals consolidation of the lung. The alveolar spaces are filled with frothy alveolar exudates (100×). (D) Periodic acid-Schiff and Grocott's methenamine silver stains reveal *Pneumocystis jirovecii* in the alveolar spaces (400×).

and linear infiltrates with or without hilar adenopathy, lung involvement from Kaposi's sarcoma should be considered [8]. With the evolving trend of immunosuppressive treatment, a high index of suspicion is advised for timely diagnosis of this potentially treatable complication among patients with connective tissue diseases. An aggressive diagnostic approach including invasive procedures is required in an immunocompromised host with unexplained pulmonary infiltrates. Early appropriate antibiotic administration enhances the chances of recovery in these patients.

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