Radiology Corner

Pneumocystis Jiroveci Pneumonia

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Note: This is the full text version of the radiology corner question published in the October 2008 issue, with the abbreviated answer in the November 2008 issue.

We present a case of Pneumocystis jiroveci (formerly P. carinii) Pneumonia (PJP). A 60 year old HIV+ male with a CD4+ count of 144 cells/mm^3 complaining of cough, dyspnea, and chest pain was ultimately diagnosed with PJP by lung biopsy after negative bronchoalveolar lavage (BAL). This case demonstrates typical radiographic findings of PJP, with symmetric bilateral lower lobe reticulonodular ground-glass opacities, complicated by spontaneous pneumomediastinum and pneumothorax.

Summary of Imaging Findings

Admission PA chest radiograph (Fig 1) reveals hypoinflated lung volumes and increased bibasilar reticulonodular ground-glass opacities. Admission contrast-enhanced CT (Fig 2) reveals diffuse multilobar ground-glass opacities with sharp transitions between normal and abnormal lung, and interstitial thickening in a predominately lower-lobe distribution.

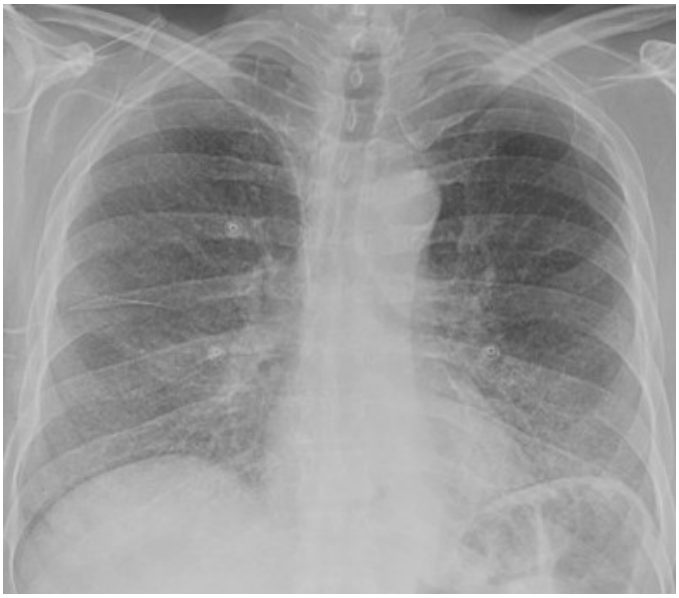

Fig. 1: Admission PA chest radiograph demonstrates bibasilar reitculonodular ground-glass opacities.

Fig. 2: Contrast-enhanced axial CT image shows ground-glass attenuation and interstitial thickening in a predominately lower-lobe distribution.

Most patients have characteristic radiographic findings of PJP, eliminating the need for CT.[1] However, in patients with pulmonary symptoms and nonspecific radiographic findings, CT often reveals typical findings, that when correlated with the CD4+ count and other clinical markers may allow the correct diagnosis of PJP to be made.[1] Other causes of ground-glass opacities, and interstitial infiltrates in patients with AIDS include CMV pneumonia, lymphocytic interstitial pneumonia, MAI infection, cryptococcal infection, Legionella, Mycoplasma, Chlamydia pneumoniae, AIDS-related lymphoma, Kaposi sarcoma, hypersensitivity pneumonia, and interstitial pulmonary edema from volume overload.

The patient underwent BAL on hospital day 3, which recovered a few red blood cells, but no organisms on gram stain or acid fast bacteria. The fluid was negative for Legionella, Streptococcus pneumoniae, and Cryptococcus neoformans. Pneumocystis direct fluorescent antibody (DFA) of the fluid was also negative. However, LDH was elevated at 506 U/L.

On hospital day 6, the patient's pulmonary status deteriorated, necessitating intubation, and the subsequent AP chest radiograph (Fig 3) reveals pneumomediastinum, with subcutaneous emphysema, and persistent bibasilar reticulonodular ground-glass opacities.

A second AP chest radiograph (Fig 4) obtained several hours later, secondary to unrelenting respiratory distress, demonstrates a significant left-sided pneumothorax (short arrows), pneumomediastinum with subcutaneous emphysema (long arrows), and persistent bibasilar reticulonodular ground-glass opacities.

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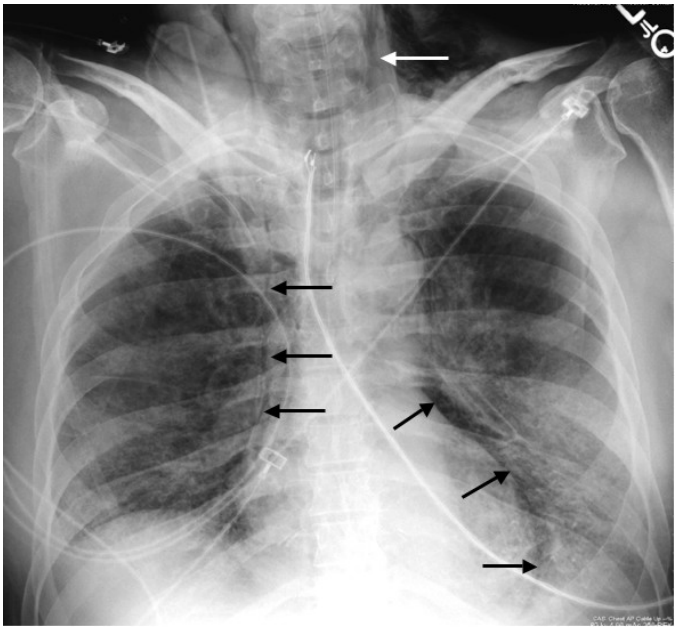

Fig 3: AP chest radiograph reveals pneumomediastinum (long black arrows), subcutaneous emphysema (long white arrow), and bibasilar reticulonodular ground-glass opacities. Note the wide lucency projecting over the left cardiac border consistent with pneumomediastinum (and/or medial pneumothorax?), in contradistinction to Mach effect.

On hospital day 10, another CT of the chest was performed (Fig 5) revealing persistent pneumomediastinum (short white arrows), left-sided anterior pneumothorax (long white arrows), a small right-sided pleural effusion (black arrows), and ground-glass opacities with focal areas of consolidation distributed throughout the bilateral lung bases and mid-lung fields.

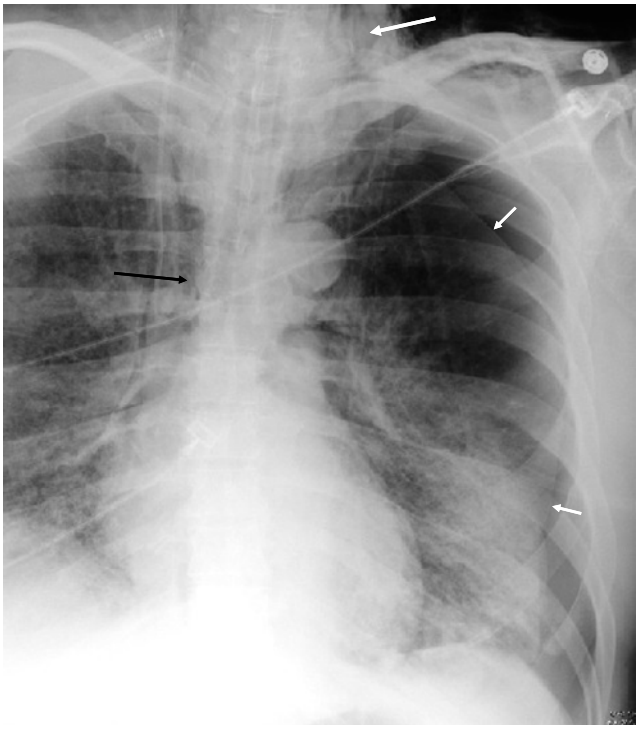

Fig 4: AP chest radiograph reveals pneumothorax (short arrows), pneumomediastinum (long black arrow), subcutaneous emphysema (long white arrow), and bibasilar reticulonodular ground-glass opacities.

Following the chest CT on the same hospital day, the patient underwent open lung biopsy of the lingula, and anterior segment of the left upper lobe. The specimens revealed marked interstitial fibrosis, focal type II pneumocyte hyperplasia, squamous metaplasia, and scattered residual alveoli consistent with a fibrosing pattern of diffuse alveolar damage. These findings are characteristic of the typical histologic findings of PCP, which consist of proliferation of type II pneumocytes, diffuse alveolar damage, granulomatous inflammation, and cyst formation.[1] Additionally, the Gomori methenamine silver (GMS) stain was positive, confirming the suspected diagnosis of Pneumocystis jiroveci pneumonia.

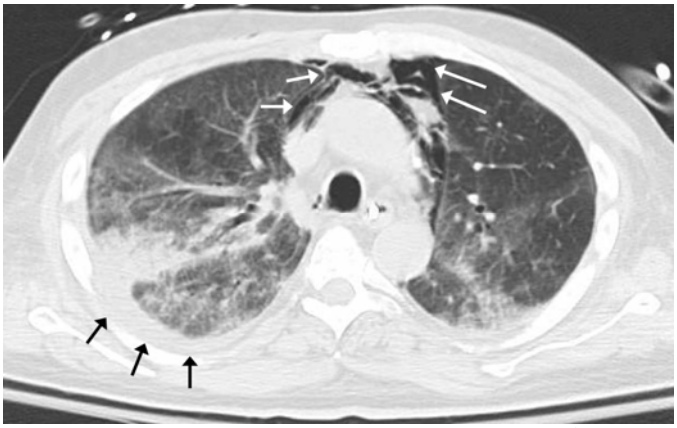

Fig 5: Contrast-enhanced axial CT image through mid-lung fields shows persistent pneumomediastinum (short white arrows), persistent left side-pneumothorax (long white arrows), right side pleural effusion (black arrows), and ground-glass attenuation with focal consolidations in bilateral lung bases.

Discussion

Pneumocystis jiroveci (P. carinii) originally thought to be protozoan, now considered a fungus, results in clinically significant pneumonia, and is an AIDS-defining illness that occurs when the CD4+ count drops to below 200 cells/mm3.[1, 2] Additionally, any immunocompromised patient may be affected; organ recipients on immunosuppressive treatment, oncologic and hematologic patients, the elderly, severely malnourished children, and patients requiring prolonged corticosteroid therapy.

The most common radiographic finding in patients with PJP consists of diffuse, bilateral symmetric reticulonodular ground-glass, or finely granular opacities occurring in a perihilar, or lower lung zone distribution.[1, 3] Less commonly, PJP will occur with upper lobe predominance.[1] If untreated, these opacities may manifest as diffuse airspace consolidation, and interstitial disease will become more pronounced, with air cysts noticed in 5% to 35% of patients.[1] Interestingly, the initial chest radiograph may be normal in as many as 39% of cases.[4]

Other radiographic findings include single or multiple nodules, miliary nodules, cavitation, hilar or mediastinal adenopathy, and pleural effusion.[1] CT typically demonstrates symmetric bilateral ground-glass opacities, which may be diffuse, or have a mosaic pattern with sharp transitions between normal and abnormal lung giving a “crazy-paving”

pattern.[1] Spontaneous pneumothorax may be identified in 5% to 10% of patients,[1,5] and is more common when cysts are present. The identification of a spontaneous pneumothorax in an HIV-positive patient is virtually diagnostic of PJP, and when visualized is a poor prognostic indicator.[4] Pneumomediastinum may also be evident occurring by itself or in concert with pneumothorax.[1]

In the above case, the presence of both pneumomediastinum and pneumothorax raises the possibility that Mach bands are responsible for the radiographic findings. Mach bands are visual phenomena caused by receptive field interactions when viewed with the juxtaposition of two nearly equal densities, making the interface appear more radiolucent. The apparent radiolucency along the edge of a structure may either enhance the perception of a small pneumomediastinum or pneumothorax, or may be mistaken for air when none is present.[6] To help differentiate between a Mach band and a pneumomediastinum/pneumothorax, one may appreciate secondary signs of intrathoracic pathology, such as air outlining the central edge of the diaphragm normally obscured by soft tissues of the mediastinum and the heart ("continuous diaphragm sign"), subcutaneous emphysema, a deep sulcus sign, or evidence of mediastinal displacement and tracheal deviation. In this case the lucency is too wide and irregular for a Mach band.

Clinically, patients with PJP demonstrate nonspecific complaints. Fever, cough, progressive dyspnea on exertion, weight loss, night sweats, and chest pain are all common symptoms.[1] Lactate dehydrogenase (LDH) levels are frequently elevated,[1, 3, 7] and patients demonstrate restrictive patterns on pulmonary function tests, with hypoxemia when exercising. [1] Clinicians may employ induced sputums, or use bronchoscopy with BAL to clinch the diagnosis. BAL, which has close to 100% sensitivity and 87% specificity in diagnosing PJP,[1, 8] is useful when positive; however, when results are negative, transbronchial or open lung biopsy may be pursued if the clinical findings suggest a high probability of PJP. Patients will often begin empirical therapy in the absence of definitive diagnosis to avoid potential complications. [1]

Summary: Perihilar and bilateral lower lobe reticular opacities on chest radiography in a patient with a CD4+ count less than 200 cells/mm^3 suggest PJP. In many cases, this is sufficient for diagnosis. However, when the chest radiograph is normal or non-specific, CT is helpful in narrowing the differential diagnosis by further defining parenchymal abnormalities. The strong radiographic evidence coupled with the patient's clinical course and negative BAL necessitated open biopsy to diagnose PJP.

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