Rapid Diagnosis of Acute Eosinophilic Pneumonia (AEP) in a Patient With Respiratory Failure Using Bronchoalveolar Lavage (BAL) With Calcofluor White (CW) Staining

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A diagnosis of exclusion, acute eosinophilic pneumonia (AEP) is an acute febrile illness with respiratory impairment, diffuse pulmonary infiltrates, and bronchoalveolar lavage (BAL) fluid eosinophilia. Whether pulmonary eosinophilia in AEP is primary or secondary remains undetermined. We report here a 22-year-old auto mechanic with severe AEP and acute respiratory failure who required intubation and ventilatory support. The patient's bronchoalveolar lavage (BAL) fluid was analyzed using cultures, cytology, Wright/Giemsa, Gram, Gomori-methenamine-silver (GMS), and calcofluor white (CW) stains (1). Despite

extensive evaluation, no infectious etiology was found. CW staining helped us rapidly to exclude *Pneumocystis carinii* or fungal infection and to focus attention toward the diagnosis of AEP. Transbronchial biopsy was unnecessary and supportive therapy without systemic glucocorticoids was followed by recovery within a few weeks. In this case, bronchoalveolar lavage with CW staining was of great assistance in the rapid diagnosis and initial management of AEP. Our literature review found no prior article using CW staining for evaluation of AEP. J. Clin. Lab. Anal. 11:202–207, 1997. © 1997 Wiley-Liss, Inc.

Key words: eosinophilic; pneumonia; calcofluor white

INTRODUCTION

Acute eosinophilic pneumonia (AEP) or “acute eosinophilic alveolitis” is an acute febrile illness with respiratory impairment, diffuse pulmonary infiltrates, and bronchoalveolar fluid eosinophilia (2–4). The pathogenesis is often unknown, but it may result from infectious, allergic, or toxic insult to the lung. AEP differs from chronic eosinophilic pneumonia in patient demographics, clinical features, and response to therapy (5–7).

We report here a 22-year-old male auto mechanic who presented to West Virginia University Hospital in acute respiratory failure requiring intubation and ventilatory support. Despite extensive evaluation, no infectious etiology for the respiratory distress was found. Broncho-alveolar lavage (BAL) with calcofluor white (CW) staining (1) helped rapidly to exclude an infectious etiology and to confirm the diagnosis of AEP.

MATERIALS AND METHODS

Broncho-alveolar lavage (BAL) was performed through the suction channel of a flexible bronchoscope with care to avoid

suctioning upper airway secretions and to minimize lidocaine instillation prior to lavage. Aliquots of nonbacteriostatic 0.9% saline at room temperature were instilled into the lung and aspirated into sterile suction traps, until ~50 ml of BAL fluid was retrieved, representing 40–60% of the instilled volume.

Using a West Virginia University laboratory algorithm for BAL fluids (1), one portion was submitted to the hematology laboratory for cell counts, differential, and rapid staining, including Wright/Giemsa, Gram, Gomori-methenamine-silver (GMS), and calcofluor white. These results were reported by the laboratory within 2 hours. Other portions were submitted for cytopathologic and microbiologic studies.

Calcofluor white (Fungi-Fluor Kit, Polysciences, Warrington, PA) is a water-soluble, colorless, nonspecific dye that selectively binds to cellulose and chitin. An ideal stain for *Pneumocystis carinii* and fungi, CW fluoresces either blue-

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white or yellow-green when exposed to ultraviolet light using an appropriate exciter-barrier-dichroic filter combination (8,9). Using a Nikon Epi-Fluorescent Optophot microscope and a 100x oil immersion objective, a blue-white image is obtained using a 330-390 nm exciter filter, 420 nm barrier filter, and Nikon UV-2A cube with dichroic mirror of 510 nm (the system we prefer) (Fig. 1A; positive control). *Pneumocystis carinii* cysts appear as white, round, uniform, nonbudding, thick-walled, 5–8 μ m spheres with intensely staining “double-parentheses” structures (10–12). Eosinophils are identified by white granule-autofluoresce on a blue background (unpublished). A yellow-green image can be obtained using a 420-490 nm exciter filter, 510-530 nm barrier filter, and Nikon B-3A cube with dichroic mirror of 510 nm (Fig. 1B; positive control).

The National Library of Medicine (MEDLINE) database was searched from 1984 onward for English or foreign language articles using the terms “eosinophilic pneumonia” or “calcofluor white.” The subset of articles or abstracts pertinent to acute eosinophilic pneumonia (AEP) or calcofluor white (CW) staining was reviewed. No prior article was identified that used CW staining for evaluation of AEP.

RESULTS

A 22-year-old white male presented with a several-day history of nonproductive cough, backache, cold sweats, and steadily worsening dyspnea. Antitussives and acetaminophen had not helped. The patient had previously been healthy, but allegedly smoked 1/2 pack-per-day cigarettes and used alcohol excessively. He had worked as an auto mechanic for the past year, had previously been a welder and coalyard worker, and frequently used solvents to clean car parts.

On admission to West Virginia University Hospital

(WVUH), the patient was extremely anxious, diaphoretic, in moderate respiratory distress, with blood pressure 112/70 mm Hg, pulse 100/min, respirations 24/min, temperature 38.6°C, and body mass index (BMI) of 26.3 kg/m². The heart was regular without murmur, gallop, or rub. The chest had diffuse bilateral rales, but no dullness to percussion, no rhonchi, or wheezes. The abdomen and extremities were unremarkable, without hepatosplenomegaly, clubbing, cyanosis, or edema.

Chest X-ray revealed prominent interstitial markings, blunting of both costophrenic angles, Kerley-B lines at both bases, and normal cardiac configuration (Fig. 2). Expecterated sputum contained very rare neutrophils and a few normal oral flora.

The admission Hgb was 17.4 g/dL and Hct was 50.4%. Red cell indices and platelet count were normal. Initially, the WBC was 21,200/microL with 82% neutrophils and 2% eosinophils; after 10 days, the WBC was 23,900/micoL with 41% neutrophils and 42% eosinophils. Admission chemistry studies were noncontributory. Serum rheumatoid factor and anti-nuclear antibody (ANA) were negative; IgA, IgM, IgD, IgE, C3, and C4 levels were normal; IgG level was slightly decreased to 623 mg/dL.

Multiple Gram and acid fast bacillus (AFB) stains of blood, urine, and sputum specimens were negative. Aerobic and anaerobic blood cultures, sputum cultures (bacteria, legionella, mycoplasma, fungi), urine cultures (bacteria, mycobacteria), viral cultures, and stool tests (ova and parasites) were all negative. Antivaricella zoster and *Toxocara canis* antibodies were in the low-positive range, suggesting past exposure to these organisms.

After cultures and serology were obtained, the patient was hydrated and received intravenous cefuroxime, trimethoprim-sulfisoxazole, erythromycin, multivitamins, thiamine, folic acid, pyridoxine, magnesium hydroxide, and cimetidine. He received albuterol and beclomethasone inhalers overnight, but

Fig. 1. Positive control BAL fluid stained with calcofluor white showing *Pneumocystis carinii* cysts and trophozoites with “double parentheses” appearance and lightly stained cell wall (magnification $\times 5000$). The image (A: left) was obtained using a 330-390 nm exciter filter, 420 nm barrier

filter, and Nikon UV-2A cube with dichroic mirror of 510 nm. The image (B: right) was obtained using a 420-490 nm exciter filter, 510-530 nm barrier filter, and Nikon B-3A cube with dichroic mirror of 510 nm.

Fig. 2. Chest radiograph (08-01-94) showing prominent interstitial markings and blunting of both costophrenic angles, but normal cardiac configuration.

did not receive systemic glucocorticoids. The morning after admission, his respiratory distress worsened, and he was transferred to an intensive care unit where he was intubated and mechanically ventilated (Fig. 3).

Bronchoscopy showed severe tracheobronchitis. Selective bronchoalveolar lavage (BAL) obtained ~50 mL of hazy, colorless fluid, which contained rare ciliated columnar epithelial cells. The fluid red cell count was 433/microL, and white cell count was 228/microL with 36% lymphocytes, 26% eosinophils, 17% macrophages, 11% monocytes, and 10% neutrophils. Multiple cytopsin preparations of the BAL fluid were

Fig. 3. Chest radiograph (08-02-94) showing worsening of the interstitial infiltrates 24 hr later, requiring placement of a right pulmonary artery catheter and endotracheal intubation.

stained by the Wright/Giemsa, Gram, and calcofluor white techniques. These studies showed a mixed inflammatory infiltrate with a predominance of lymphocytes and eosinophils (Fig. 4). Occasional reactive lymphocytes were seen. No bacteria were identified, and there were no evident fungi, viral inclusions, "foamy alveolar" casts, cysts, or trophoblasts. Cytospin preparations sent for AFB and GMS stains were negative. Fluid cytology was negative for malignant cells, *Pneumocystis carinii*, fungal forms, acid fast bacilli, and viral cytopathic effect. These findings were considered most consistent with a noninfectious pulmonary process.

With supportive care, the patient gradually improved and his chest radiographs and peripheral blood eosinophilia improved. He was extubated on the seventh hospital day, and was discharged on the thirteenth day without glucocorticoids or other medications. Final diagnosis was "acute respiratory failure secondary to acute eosinophilic pneumonia—eosinophilic alveolitis." The etiology of the AEP and his long-term prognosis were considered unknown, since exposure to unidentified environmental toxins or allergens could not be excluded.

DISCUSSION

In 1989, Allen et al. reported four patients with acute eosinophilic pneumonia (AEP). This entity was considered to be an acute febrile illness of <7 days duration with severe hypoxemia, diffuse pulmonary infiltrates, increased eosinophils in bronchoalveolar-lavage fluid, no pulmonary or systemic infection, and no atopic illness (2–4). Other authors subsequently defined AEP as a respiratory illness with symptoms < 1-month, acute onset with high fever, diffuse ground-glass and micronodular infiltrates, interstitial pulmonary edema (13,14), bilateral pleural effusions, or mild mediastinal lymphadenopathy (15), spontaneous improvement, and no relapses (5–7,16).

Fig. 4. Wright-Giemsa stained smear showing bronchoalveolar lavage (BAL) fluid eosinophilia (08-02-94; magnification ×1500).

Pathologic studies in AEP have shown enlarged mediastinal lymph nodes with sinus histiocytosis, sinus eosinophilia, and compression of lymphoid follicles (17). Lung biopsies have shown infiltration of bronchial wall mucosa by activated, degranulated eosinophils, which are also present in alveolar spaces, in thickened alveolar septae, and in perivascular areas (5,18). Pulmonary eosinophils have appeared hypodense with nuclear hypersegmentation (19).

Eosinophilic pneumoniae have multiple etiologies (Table 1) and overlapping clinical presentations and findings (20). AEP has been associated with inhalation of Scotchguard (1,1,1-trichloroethane) (21), heroin (22), or stainless steel dust containing 0.1% nickel (23); with exposure to red spider allergens (24) or radiographic contrast (25); with infection with human immunodeficiency virus (26), *Trichosporon terrestris*

(27), *Toxocara canis* (28,29), *Trichosporon cutaneum* (30), *Trichoderma viridae* (30,31), *Aspergillus* species (30,32), *Mycobacterium tuberculosis* (33), Coxsackie A2 virus, or *Pseudomonas maltophilia* (32); with treatment with acetaminophen (34), bleomycin (35), loxoprofen (36), minocycline (37-40), naproxen (41), nitrofurantoin (42), pentamidine (43), salazopyrin (44), sulfadoxine (45), sulfasalazine (46,47), or tolfenamic acid (48). The current patient smoked cigarettes, as did up to 80% of AEP patients in prior reports (3,4,7,16).

Since significant alveolar eosinophilia occurs infrequently, and different etiologies have different therapeutic implications, one should attempt to establish the specific etiology in all patients. Bronchoalveolar lavage (BAL) has proven extremely useful for rapid diagnosis of eosinophilic pneumoniae, pulmonary infections, or neoplasms (49,50).

BAL fluid normally contains ~90% alveolar macrophages and ~10% lymphocytes with only occasional neutrophils, eosinophils, or basophils (51). In one large series, 95% of normal persons had < 5% alveolar eosinophils (3); other authors have considered > 10% alveolar eosinophils to be abnormal (52,53). Alveolar eosinophilia usually occurs during acute eosinophilic pneumonia, interstitial lung diseases, acquired immunodeficiency syndrome associated pneumonia, idiopathic eosinophilic pneumonia, or drug-induced lung disease (3), but is uncommon during adult respiratory distress syndrome, lung cancer, community-acquired pneumonia, or in immunocompromised persons without AIDS (3).

Calcofluor white staining of BAL fluids equals or exceeds the sensitivity of silver stains for rapid identification of opportunistic respiratory pathogens (1,11,12,54-62). CW staining is both rapidly performed and easily interpreted, although experience in ultraviolet fluorescent microscopy is necessary. In our hands, results are available in < 2 hr, allowing better selection of subsequent virology cell cultures, electron microscopy, and other assays. Laboratory costs for each BAL sample are reduced by ~30%, and the methodology is available 7 days weekly (1).

Although patients with AEP may improve spontaneously without corticosteroids (63), severe respiratory failure (64) and death (65) have been reported. Many clinicians favor administering corticosteroids after examination of lavage-fluid stains and before the final results of cultures are available (3,4). In the current patient, BAL with calcofluor white staining was an extremely useful adjunct for rapidly excluding infectious etiologies and narrowing the differential diagnosis, allowing us to use conservative, supportive therapy without systemic glucocorticoids.

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TABLE 1. Causes of Eosinophilic Pneumonia

Drugs
Antibiotics (e.g., nitrofurantoin, penicillin, sulfonamides)
Antineoplastics (e.g., methotrexate, tamoxifen)
Antiinflammatory agents (e.g., aspirin, gold, naproxen)
Antiarrhythmics (e.g., quinidine)
Antiasthmatic agents (e.g., cromolyn sodium)
Antihyperglycemics (e.g., chlorpropamide, tolbutamide)
Antihypertensives (e.g., hydralazine)
CNS-altering agents (e.g., chlorpromazine, carbamazepine)
L-tryptophan (eosinophilia-myalgia syndrome)
Muscle relaxants (e.g., dantrolene)
Hypereosinophilic syndrome
Cardiomyopathy with hydrostatic pulmonary edema
Pulmonary parenchymal infiltration
Idiopathic
Acute eosinophilic pneumonia
Chronic eosinophilic pneumonia
(Cigarette smoking ?)
Interstitial lung disease
Pulmonary hemosiderosis
Simple pulmonary eosinophilia (Loeffler's)
Immunologic diseases
Rheumatoid diseases
Sarcoidosis
Infections
Bacterial (e.g., <i>Brucellosis</i>)
Fungal (e.g., <i>Coccidioidomycosis</i> , AIDS associated <i>Pneumocystis</i>)
Viral "pertussoid" eosinophilic pneumonia
Nematodes (filaria, <i>Toxocara canis</i> , <i>Ascaris lumbricoides</i>)
Inflammatory airway diseases
Bronchocentric granulomatosis
Bronchial asthma with mucoid impaction ("Chronic eosinophilic bronchitis")
Hypersensitivity pneumonitis (<i>Aspergillus</i> , <i>Micropolyspora</i> , <i>Thermoactinomyces</i> , etc.)
Neoplasia
Carcinoma (e.g., bronchogenic)
Immunoblastic lymphadenopathy
Lymphoma (e.g., Hodgkin's Disease)
Vasculitides
Allergic angiitis and granulomatosis (Churg-Strauss)
Polyarteritis nodosa
Wegener's granulomatosis

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