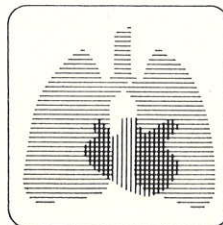


Mucor Mediastinitis*

Bradley A. Connor, M.D.;^{,*} Ron J. Anderson, M.D.;[†] and
James W. Smith, M.D.[§]*



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Mucor Mediastinitis*

*Bradley A. Connor, M.D.,** Ron J. Anderson, M.D.,† and James W. Smith, M.D.‡*

A 69-year-old man with lymphocytic leukemia presented with fever, a pericardial friction rub, widening of the mediastinum, and left pleural effusion. Atrial fibrillation, refractory hypotension and acute paraplegia punctuated his hospital course. Invasion of the mediastinum, myocardium, mediastinal, coronary and spinal arteries with mucormycosis was present at post-mortem examination.

Mediastinitis usually occurs secondary to infection involving structures passing through the mediastinum.¹ Perforation of the esophagus or pharynx, retro-

*From the Department of Internal Medicine, University of Texas Health Science Center at Dallas, and Medical Service, Veterans Administration Hospital, Dallas.

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†Chief, Ambulatory Care, Parkland Memorial Hospital; Assistant Professor of Internal Medicine, University of Texas Health Science Center at Dallas.

‡Chief, Infectious Diseases, Dallas VA Hospital; Associate Professor of Internal Medicine.

Reprint requests: Dr. Anderson, UTHSC at Dallas, 5323 Harry Hines Blvd, Dallas 75235

pharyngeal abscess, facial cellulitis, contiguous pulmonary or pericardial infection can each lead to acute suppurative mediastinitis.^{2,3} We report spread of pulmonary mucormycosis to the mediastinum in an immunosuppressed patient.

CASE REPORT

The patient, a 69-year-old white man with known chronic lymphocytic leukemia for one year, returned to the Oncology Clinic complaining of generalized malaise. He was admitted for evaluation of thrombocytopenia (platelet count 69,000/cu mm). His current medications included prednisone 80 mg per day, and cyclophosphamide (Cytosan) 100 mg/day.

On physical examination, his vital signs revealed a pulse rate of 96, temperature 98.6°F (37.5°C), blood pressure 128/80 mm Hg, and respirations 20 per minute. The patient had developed a high pitched, hoarse voice in addition to dullness to percussion in the left lung base and bilateral rales. The heart was moderately enlarged with a PMI 3 cm lateral to the midclavicular line. A grade 2/6 systolic ejection murmur at the apex and a three-component friction rub in the third left intercostal space were heard. No peripheral edema was present. The tip of the spleen was easily palpable and the patient had normal findings on neurologic examination. Frequent premature ventricular beats were noted by electrocardiogram and the admission chest x-ray film showed "borderline cardiomegaly unchanged from previous examination." The lung fields were free of infiltrates.

After admission, the patient's temperature rose to 102°F (38.8°C) and quickly defervesced. Appropriate material for cultures was obtained. A repeat chest x-ray film was obtained which suggested a slight increase in the cardiac silhouette. Shortly thereafter, the patient developed atrial fibrillation with a ventricular response of 160, the blood pressure dropped from 140/90 to 96/78 mm Hg, and an ill-defined paradox (8 to 15 mm Hg) was evident in the face of central venous pressure of 7 cm water. Rapid intravenous administration of digitalis was successful in controlling the

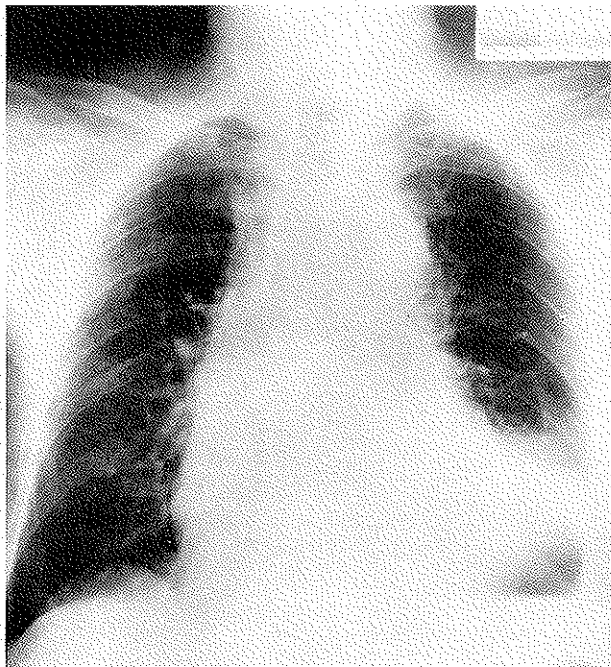


FIGURE 1. Chest x-ray film just prior to aortography.

patient's heart rate. An echocardiogram was negative for pericardial effusion.

On the second hospital day, the patient again was febrile to 101.6°F (38.7°C). Another chest x-ray film revealed a left lower lobe infiltrate, moderate-sized left pleural effusion, small right pleural effusion, and definite mediastinal widening (Fig 1). Pleural fluid examination revealed 2.5 gm percent protein, 1,500 white blood cells (61 percent polys), 600 red blood cells, there were no acid-fast bacteria and fungal smears were negative. Despite negative cultures and serologic tests drawn earlier, intravenous administration of penicillin and gentamicin was started empirically. Because of the widening mediastinum, emergency aortography was performed to rule out an aortic dissection. This study gave negative findings except that the left diaphragm was noted not to move during fluoroscopic examination.

On the evening of the fourth hospital day, the patient complained of sudden inability to move his lower extremities. Neurologic examination defined a paraplegia localized at the T10-T11 level. Lumbar puncture revealed an opening pressure of 12 cm H₂O, cerebrospinal fluid glucose 94/serum glucose 289, white blood cell count 186 (72 percent polys), red blood cell count 70, protein 80 mg percent. Gram stain, india ink preparation and smears for acid-fast bacteria were negative.

The patient remained febrile to 102°F (38.8°C), developing labored respirations on the fifth hospital day. Chest x-ray film revealed a white left lung. Repeat examination of cerebrospinal fluid was remarkable for a glucose of 19/serum glucose 321, white blood cell count 740 (93 percent polys), and 258 mg percent protein. Early on the morning of the

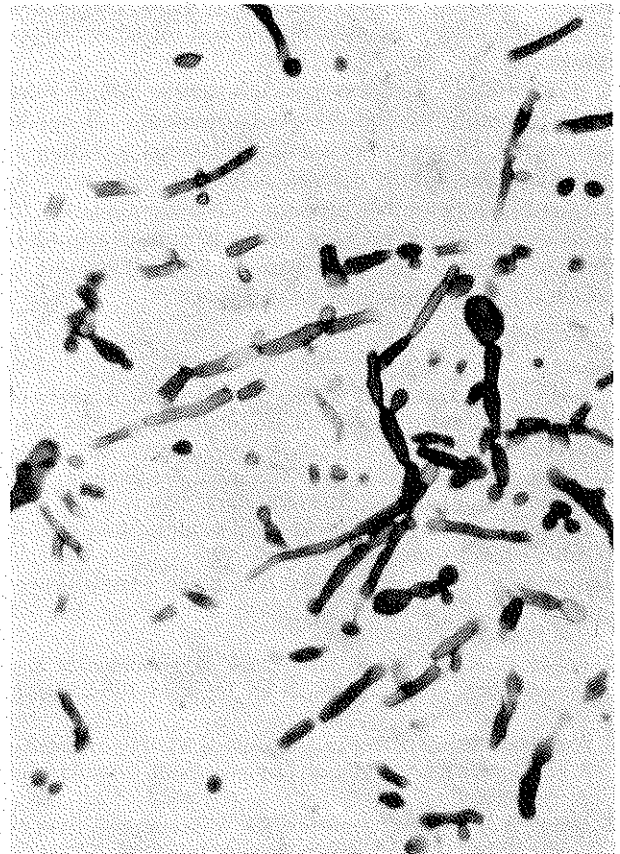


FIGURE 2. Methenamine silver stain of culture from mediastinum illustrating broad nonseptate hyphae characteristic of *Mucor* species.

sixth hospital day, refractory hypotension developed followed by respiratory arrest from which the patient could not be resuscitated.

At autopsy, massively enlarged lymph nodes were noted in the mediastinum. A large, firm, brownish-yellow mass was attached to the lingula of the left lung extending into the mediastinum. A prominent fibrinous pericarditis was present with only 10 ml of a clear fluid. The left pleural cavity contained a dense, fibrous adhesion with 650 ml of straw-colored fluid. The aorta was constricted just below the arch to a diameter of 1.5 cm by the mass. The spinal canal contained several areas of a nodular material at T12. Microscopic examination of this material revealed broad nonseptate hyphae invading spinal blood vessels. Hyphae were also found in mediastinal arteries and in the anterior descending branch of the left coronary artery resulting in a myocardial infarction. Fungal cultures of this material grew *Mucor* species (Fig 2). No evidence of chronic lymphocytic leukemia was found and it is assumed that the patient was in complete remission.

COMMENT

Mucorales, members of the Phycomycetes class, have been recognized to cause rhinocerebral infections in diabetics with ketoacidosis, and rarely to produce pulmonary infarction in the diabetic patient.⁴⁻⁶ Aggressive chemotherapy which leads to leukopenia in patients with leukemia or lymphoma may predispose to mucormycosis.⁷ The hallmark of this infection is vascular invasion by characteristic broad nonseptate hyphae. In this patient, vascular invasion resulted in acute paraplegia, myocardial infarction, arrhythmias and refractory hypotension.

It is unlikely that this patient's course would have been favorably altered by mediastinoscopy or mediastinotomy; however, fever and mediastinal widening of a nonvascular nature deserve definitive procedures to rule out a suppurative process. Early identification of *Mucor* species by impression smears or culture (requires three to four days) may allow amphotericin B therapy to which these organisms may be sensitive.⁸

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