revealed hemorrhagic fibrinous debris with focal clustering of neutrophils and clusters of Gram-positive and poorly stained cocci consistent with infective mural endocarditis. Fungal staining was negative.

**DISCUSSION**

We present the case of a woman who had previously unrecognized mitral regurgitation. She developed an acute febrile illness and blood cultures were positive for *S aureus*. A vegetation, which had partially embolized to her upper and lower extremities and brain, was evident on echocardiography. At operation, left atrial mural endocarditis with otherwise normal-appearing cardiac anatomy was found.

Previously reported episodes of lone bacterial mural endocarditis have usually been associated with underlying disease processes ranging from thrombophlebitis to bronchiectasis and paraspinal abscess. In some of these cases, the pathogenesis of mural involvement has been ascribed to direct extension of myocardial abscesses; however, the etiology in others remained obscure. Mural endocarditis has been reported in the setting of infected mural thrombi or aneurysms, jet lesions from ventricular septal defects and idiopathic hypertrophic subaortic stenosis. In addition, left atrial mural endocarditis may be acquired from the extension of a pulmonary abscess through a pulmonary vein.

According to recent reviews, fungal endocarditis confined to the mural surface of normal hearts is observed with immunosuppression from either lymphoproliferative disorders and their treatment or organ transplantation immunomodulation. Postulate that previously undetected mitral regurgitation may have created a jet lesion on the posterior left atrial wall, creating an anatomic substrate for infection.

*Staphylococcus aureus* is an unusual pathogen in native valve infective endocarditis, with only an overall 1.5 to 13 percent prevalence in recent review. This virulent organism usually lodges itself on normal valves and is more frequent in intravenous drug abusers.

M-mode and two-dimensional echocardiography have been of diagnostic and prognostic value in bacterial and fungal endocarditis. The echocardiographic findings in this patient were striking, with the vegetation prolapsing into the left ventricle during diastole, only to recede into the left atrium during systole, mimicking a myxoma. Although 75 to 80 percent of myxomas are found in the left atrium, they usually are attached to the limbus of the fossa ovalis by a short fibrovascular stalk and only rarely present as extremely fragile papillary excrescences which have a sessile attachment to the interatrial septum or posterior atrial wall. Superinfection and embolism are well recognized complications of atrial myxomas and though our patient did not manifest any evidence of hemodynamic obstruction, the preoperative diagnosis was an infected myxoma of the posterior left atrial wall with emboli.

It is important to recognize that the absence of vegetation on two-dimensional echocardiogram does not rule out the diagnosis of endocarditis. Fifteen percent of infective vegetations can be missed by echocardiography and false-negative results up to 50 percent have been reported in a small series of cases of *Aspergillus* endocarditis.

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**Subcutaneous and Mediastinal Emphysema Associated with Hypersensitivity Pneumonitis**

Yoichiro Ichikawa, M.D.; Naoto Tokunaga, M.D.; Masaharu Kinoshita, M.D.; Toru Rikimaru, M.D.; and Masao Kaji, M.D.

We report a rare case of a patient in whom severe subcutaneous and mediastinal emphysema occurred in association with summer-type hypersensitivity pneumonitis and in whom overdositation or disruption of alveoli with obliteration of the respiratory bronchioles was revealed on open lung biopsy. This case suggests that obstructive bronchiolitis with hypersensitivity pneumonitis is an etiologic factor of mediastinal emphysema.

(Chest 1991; 99:759-61)

Spontaneous occurrence is the most common mechanism in some etiologies and pathogeneeses of mediastinal emphysema. Spontaneous subcutaneous and mediastinal emphysema have been described as occurring under various clinical conditions, and it is common in neonates but rare in adults.

We report a rare case of a patient in whom marked subcutaneous and mediastinal emphysema occurred in association with summer-type hypersensitivity pneumonitis and we discuss the mechanism underlying the mediastinal emphysema in our patient. Summer-type hypersensitivity pneumonitis, the most prevalent form of hypersensitivity

*From the First Department of Internal Medicine, Kurume University Medical School, Kurume, Fukuoka, Japan.

Reprint requests: Dr. Ichikawa, 1st Department of Internal Medicine, Kurume University School of Medicine, 67 Asahi-machi, Kurume 830, Japan*
pneumonitis in Japan, is characterized by the following clinical features: (1) initiation in the summer and seasonal episodes; (2) familial occurrence; and (3) positive result of returning-home provocation test. In 1984, Shimazu et al identified *Trichosporon cutaneum* as an important etiologic agent of the disease.

**CASE REPORT**

A 40-year-old woman was admitted to our hospital with the chief complaint of retrosternal pain and dyspnea. Two months earlier, she had been admitted to another hospital with the complaint of low-grade fever, severe nonproductive cough that worsened at night, and exertional dyspnea. At the time, she was diagnosed as having a common cold, but her condition did not improve with the treatment of oral antibiotics. She had noted a worsening of cough and exertional dyspnea from one week before admission to our hospital. Preceded by a coughing spasm, retrosternal pain radiating to the shoulders and dyspnea at rest developed abruptly four days later. Her medical history indicated nothing of note.

On physical examination, she was dyspneic at rest, body temperature was 36.3°C, pulse rate was 96 beats per minute and regular, blood pressure was 110/70 mm Hg, and respirations were 30/min. Subcutaneous emphysema was revealed in her right cheek, neck, shoulders, and anterior chest wall. Crepitations were detected on palpation. Crunching (Hamman’s sign) was noted on auscultation over the chest. Her white blood cell count was 9,700/cu mm with 67 percent neutrophils, 32 percent lymphocytes, and 1 percent eosinophils. Electrolytes and liver function test results were normal. The patient’s purified protein derivative was negative. Her serum angiotensin-converting enzyme level was normal. The indirect fluorescent antibody titer to *T. cutaneum* of her serum was 1:32. Antibodies to the other fungal antigens and to pigeon serum were not detected.

Arterial blood gas in room air showed a pH of 7.47, PCO2 of 35 mm Hg, and PO2 of 51 mm Hg. Spirometry revealed a moderately restrictive defect with a VC of 2.0 L, a VC/VC predicted rate of 72.2 percent, an FEV1 of 1.85 L, an FEV1/FVC of 88 percent, a TLC of 3.98 L, and a RV/TLC of 45.7 percent. Her RV was 1.82 L, FRC was 3.1 L, and Dco was 55.7 percent of predicted values.

The chest roentgenogram (Fig 1) revealed gas shadows that were identified along the heart border and muscles of the neck with diffusely disseminated fine granular shadows in both lower lung fields. Gallium-67 scanning showed a marked accumulation in both lung fields.

Bronchoalveolar lavage (BAL) was performed in the right middle lobe segment, and cellular analysis of the lavage fluid revealed 22 percent alveolar macrophages, 44 percent lymphocytes, 8 percent neutrophils, and 26 percent eosinophils. Lymphocytes recovered by BAL consisted of 36 percent helper T cells (CD4) and 46.5 percent suppressor T cells (CD8). The CD4/CD8 ratio was 0.77.

Open lung biopsy specimens demonstrated epithelioid cell granulomas in the peribronchiolar area with mild alveolitis. The granulomas consisted of infiltration with lymphocytes, plasma cells, and giant cells of both Langhans and foreign body types. This granulomatous inflammation resulted in obstruction or narrowing of respiratory bronchioles, and the surrounding alveoli were emphysematous with destruction of their walls. These pathologic findings were compatible with hypersensitivity pneumonitis (Fig 2).

The patient made spontaneously favorable progress after hospital admission. On the 40th hospital day, she was free of pulmonary symptoms except for a slight cough with normal chest roentgenogram and PO2 level.

**DISCUSSION**

The mechanism behind mediastinal emphysema is rupture of the marginally situated alveoli resulting from a marked increase in intra-alveolar pressure or a decrease in perivascular interstitial pressure or both. Increased alveolar pressure commonly occurs with obstructed expiratory airflow. Extra-alveolar air following rupture of alveoli dissect along the bronchovascular sheaths to the lung hilum and mediastinal soft tissues, and then escapes from the mediastinum along the great vessels into the neck and chest wall.

The common causes of spontaneous subcutaneous and mediastinal emphysema have been reported to be Valsalva's manoeuver, violent cough, emesis, acute bronchial asthma, assisted ventilation of respiratory distress in infants, pneumonia associated with viral infection such as measles, and others. Macklin and Macklin emphasized the predisposition to alveolar disruption when there is preexisting infiltration, either infective or inflammatory, of the pulmonary

![Figure 1.](image1.png) The chest roentgenogram demonstrated longitudinal gas shadows that were identified along the heart border and in the subcutaneous soft tissues of neck and chest wall with disseminated fine granular shadows in both lower lung fields.

![Figure 2.](image2.png) Photomicrograph of the open lung biopsy specimen showed disseminated epithelioid cell granulomas, including giant cells of both Langhans and foreign body types in peribronchiolar area, and obstructive bronchiolitis with overdistention or disruption of surrounding alveoli (hematoxylin-eosin, original magnification x 50).
parenchyma. However, few reports have appeared of mediastinal emphysema associated with hypersensitivity pneumonitis. In hypersensitivity pneumonitis such as farmer's lung\(^6\) or summer-type hypersensitivity pneumonitis,\(^4\) common symptoms are chronic cough, dyspnea, and fever, but mediastinal emphysema is a very rare complication.

In both acute and chronic stages of farmer's lung, obstructive bronchial lesions (obstructive bronchiolitis) are common pathologic features.\(^5\) In our patient, the open lung biopsy specimens demonstrated obstruction or narrowing of respiratory bronchioles resulting from development of numerous epithelioid cell granulomas in the peribronchiolar area and overdistention or disruption of the surrounding alveoli. In children, bronchiolitis-caused viral infection is considered to be an etiologic factor of mediastinal emphysema.\(^6\)

On the basis of these pathologic conditions, we postulate that in this patient, obstructive bronchiolitis associated with granulomatous alveolitis assumed an important role in the development of mediastinal emphysema in addition to the coughing spasm, \(ie\), rupture of alveoli resulted from increased intra-alveolar pressure probably occurring as a result of check valve bronchial obstruction secondary to bronchiolitis. From this pathologic perspective, it would seem that the occurrence of mediastinal emphysema associated with hypersensitivity pneumonitis is not so rare.

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Use of Indium 111-Labeled White Blood Cell Scan in the Diagnosis of Cytomegalovirus Pneumonia in a Renal Transplant Recipient with a Normal Chest Roentgenogram*

Kenneth Chinsky, M.D.; and Daniel M. Goodenberger, M.D., F.C.C.P.

Opportunistic infections are common in patients after renal transplantation. This report describes a case of cytomegalovirus pneumonia in a renal transplant recipient with a normal chest roentgenogram and normal arterial oxygenation. An abnormal "In-white blood cell scan led to the discovery of a pulmonary source of his recurrent fevers.

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\textbf{BAL} = bronchoalveolar lavage; \textbf{CMV} = cytomegalovirus; \textbf{HSV} = herpes simplex virus; \textbf{"In-WBC} scan = indium 111-labeled white blood cell scan \\
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Cytomegalovirus (CMV) infections are a major cause of morbidity and mortality in patients after renal transplantation. The average incidence of infection from multiple studies is approximately 71 percent.\(^1\) In one study, 20 percent of 59 hospitalized patients died as a direct result of CMV disease.\(^2\) Cytomegalovirus is the most common cause of pneumonia in renal transplant recipients,\(^3\) usually occurring one to four months after transplantation.\(^4\) Patients typically present with fever, dyspnea, hypoxemia, and an abnormal chest roentgenogram most often showing interstitial or alveolar infiltrates, although cavities and nodules have also been described.\(^5\) Lobar consolidation is rare, although this has been reported in two cardiac transplant patients from whom CMV was the only pathogen recovered.\(^6\) A definitive diagnosis of CMV pneumonitis is made by culture of the virus from bronchial washings or bronchoalveolar lavage (BAL) fluid in association with identification of cytopathic cells in washings or BAL fluid or the typical pathologic changes in tissue specimens.

Although there have been reports of CMV pneumonia occurring in renal transplant recipients with normal chest roentgenograms,\(^7\) histologic confirmation is generally absent. In one such reference,\(^8\) the diagnosis was based on a fourfold rise in antibodies against CMV early and late antigens by enzyme-linked immunosorbent assay in conjunction with a decrease in Krogh's coefficient on pulmonary function testing.

We describe a case of biopsy specimen–confirmed CMV pneumonia in a renal transplant recipient with a normal chest roentgenogram. The diagnosis was initially suspected because of an abnormal indium 111-labeled white blood cell scan ("In-WBC scan).

CASE REPORT

A 33-year-old man with end-stage renal disease due to chronic pyelonephritis underwent bilateral nephrectomy and living related donor renal transplantation in 1975. Four episodes of rejection

*From the Respiratory and Critical Care Division, Washington University School of Medicine, St. Louis.
†Pulmonary Fellow.
‡Assistant Professor of Medicine.