a ventricular septal defect and the diagnosis was confirmed by echocardiography. Accurate diagnosis of aortopulmonary window is extremely difficult on clinical grounds when it coexists with a ventricular septal defect. The heart murmur of this anomaly is often mistaken for the murmur of a high ventricular septal defect, because although a continuous murmur may be present in patients with aortopulmonary window, more often there is only a systolic murmur that is generally heard along the upper left sternal border. In this case, there was no continuous murmur because there was little left-to-right diastolic flow through the aortopulmonary window, due to the elevated pulmonary arterial diastolic pressure and to the large size of the communication.

The other cardiac anomaly that can mimic the findings in this case is a large ventricular septal defect associated with patent ductus arteriosus. Aortopulmonary window can be distinguished from patent ductus arteriosus by the detection of diastolic flow reversal proximal to the aortic isthmus using pulsed Doppler echocardiography.*

In recent years, two-dimensional echocardiography has been found to be extremely helpful in the noninvasive diagnosis of various conotruncal anomalies. In many normal individuals (especially infants), the contiguous proximal aortic and pulmonary arterial walls are thin and many appear as a false dropout on two-dimensional echocardiography. Therefore, this could potentially result in a false diagnosis of aortopulmonary window. However, Doppler color flow imaging can verify the presence of a defect by displaying evidence of flow across the echo-free space into the main pulmonary artery from the aorta.** In this case, two-dimensional echocardiography showed a large communication between the aortic arch and the main pulmonary trunk, and Doppler color flow imaging clearly indicated the flow between the aorta and the pulmonary artery. A large ventricular septal defect was also confirmed by both two-dimensional echocardiography and Doppler color flow imaging. Thus, the combination of two-dimensional echocardiography and Doppler color flow imaging as well as pulsed Doppler echocardiography can rule out other diseases that mimic or are associated with aortopulmonary window.

REFERENCES

Fibrosing Alveolitis Responsive to Corticosteroids following Legionnaires’ Disease Pneumonia*

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Two male patients ages 54 and 58 years had persisting pneumonia with dry cough, dyspnea, weight loss, and fever up to 39°C that did not respond to erythromycin treatment. There was extensive restrictive impairment of ventilation and loss of diffusing capacity for carbon monoxide. Histologic examination of the basal pulmonary infiltrates showed fibrosing alveolitis. Serologic titers indicated that the patients had suffered from Legionella pneumophila infection. We believe that Legionella had caused the fibrosing alveolitis since there was absence of any other causative agents

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Clinical interest in pneumonia caused by Legionella was aroused by the epidemic outbreak in 1976 at a meeting of war veterans and the identification of the pathogen. \(^1\) Legionella pneumophila is most commonly found of the many species. Mortality has varied between 16 percent\(^1\) and 25 percent.\(^4\)

The clinical course of the disease is mostly that of an acute infection.\(^1\) The pathologic findings are those of alveolar bacterial pneumonia.\(^1\) In some cases, prolonged courses have been observed with resultant pulmonary fibrosis leading to severe impairment of lung function or even death.\(^6\)

We herein report two patients with serologically proven Legnionnaire's disease first diagnosed 6 and 13 weeks after the onset of symptoms. Both showed evidence of fibrosing alveolitis that responded to corticosteroid treatment.

**CASE REPORTS**

**CASE 1**

A 54-year-old man was admitted to the Pulmonary Unit in October 1987 having had temperatures around 39°C for a month until the hospital admission. He had weight loss of 10 kg, dry cough, breathlessness on exertion, and night sweats. Physical examination revealed crackles over the posterior basal right lung. Chest roentgenograms demonstrated bilateral basal infiltrates (Fig 1). Laboratory examinations showed a hemoglobin concentration of 12.5 g/dl, 15.5 x 10\(^9\)/L leukocytes, erythrocyte sedimentation rate 120 mm/h, and PaO\(_2\) of 8.5 kPa. There was restrictive ventilatory impairment (FVC, 2.200 ml [predicted value 4.170 ml]; FEV\(_1\), 1.850 ml [predicted value, 2.990 ml]). The single breath lung diffusing capacity for carbon monoxide was reduced to 9.5 ml x s\(^-1\) x kPa\(^{-1}\) (predicted value, 20.7 ± 4.3).

*Legionella pneumophila* antibodies were not detected two weeks prior to hospital admission, but three weeks later the titer was 1:512 (indirect immunofluorescent test, serogroup 1-6, BISO, Munich). Allergic alveolitis, autoimmune diseases, or nontuberculous infections were excluded by serologic tests. Fibrosing alveolitis without bronchiolitis obliterans was diagnosed by transbronchial biopsy specimen. No pathogenic bacteria were found in the bronchial secretions. There was negative therapeutic response to erythromycin given for one week in a dose of 3 g daily. The patient was therefore treated with an oral dose of 60 mg of prednisone per day, which resulted in a rapid improvement of clinical symptoms. Four weeks later, both FVC and FEV\(_1\) had increased (FVC, 3.250 ml; FEV\(_1\), 2.950 ml). Three months after the onset of therapy, spirometric values were within normal limits and lung infiltrations had completely cleared. The Legionella antibody titer decreased under steroid treatment to 1:256 (three weeks), 1:128 (six weeks), and 1:64 (eight months). Five weeks after the end of the eight-month therapy, there was radiologic evidence of an asymptomatic recurrence of lung infiltrates accompanied by a raised antibody titer (1:128). Further treatment with oral prednisone again led to complete suppression of the infiltrates after two months.

**CASE 2**

In May 1989, a 58-year-old man was admitted to the hospital with fever of 39°C, that had been present for three months. He had dyspnea on exertion, a cough with yellow sputum, and a weight loss of 4 kg. Physical examination revealed acrocyanosis and crackles over both lung bases. A reticulonodular infiltration was radiologically evident in both lower lobes. Computed tomography revealed mediastinal lymph node enlargement and interstitial lung disease. Laboratory examinations showed a hemoglobin concentration of 12.5 g/dl, 6.7 x 10\(^9\)/L leukocytes, erythrocyte sedimentation rate 82 mm/h, and PaO\(_2\) of 8.2 kPa. He had restrictive ventilatory impairment (FVC, 3,000 ml [predicted value, 4.020 ml]; FEV\(_1\), 2,300 ml [predicted value, 2.820 ml]). The lung diffusing capacity for carbon monoxide was decreased to 61 percent of the normal. The \(L\) pneumophila antibody titer was initially 1:512. Other causative agents of interstitial lung disease were excluded serologically. A transbronchial lung biopsy specimen documented the presence of fibrosing alveolitis without bronchiolitis obliterans (Fig 2). Only one small group of rod-shaped bacteria was demonstrated with the Dieterle silver impregnation method. The sputum and bronchial secretions were sterile. Erythromycin therapy in a daily dose of 3 g orally for one week had no effect on the patient's illness. He was...
then treated with 50 mg of prednisone orally for four weeks and reduced dosages afterwards, which led to a dramatic improvement of symptoms. Four months after the start of therapy, FVC, FEV₁, and PaO₂ were normal, but incomplete radiologic regression was observed at this time. The antibody titer decreased to less than 1:64 after seven weeks of steroid therapy and remained below that level.

**DISCUSSION**

Acute Legionella pneumonia is a severe bacterial infection with a high mortality rate. 1,3 Fatal outcome after the acute phase subsides is not infrequent despite adequate erythromycin therapy. 4 The most prominent pathologic finding in these instances was pulmonary fibrosis. Milder courses have shown persisting alveolitis with restrictive impairment of ventilation. We believe that our patients belong to the latter group.

Infection with *L. pneumophila* was diagnosed on the basis of an indirect immunofluorescent test. In patient 1, the titer rose to 1:512, whereas in patient 2 the maximum of 1:512 was found initially. The decrease of the titers in the later stages confirmed the diagnosis. In one patient, infection was further substantiated by direct staining of bacteria with the Dieterle silver impregnation method.

The patients' chest roentgenograms and pulmonary function tests were characteristic of alveolitis for a number of months. In addition, both patients had alveolitis with interstitial fibrosis on lung biopsy specimen. The therapy with corticosteroids resulted in a dramatic improvement of clinical symptoms and lung function parameters. However, residual infiltrates were still detectable on chest roentgenograms for many months. In patients such as ours with a highly suspected causal relationship between Legionella infection and fibrosing alveolitis, we recommend corticosteroid treatment, since fatalities from lung fibrosis have been reported when treatment has consisted of erythromycin alone. 5,7

**REFERENCES**


**Primary Endobronchial Actinomycosis in Association with Foreign Body Aspiration**

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A 66-year-old diabetic man presented with a bilobar pneumonia two months after aspiration of a chicken bone. Flexible fiberoptic bronchoscopy demonstrated a mass in the bronchus intermedius. Histologic examination of endobronchial biopsy specimens revealed bone fragments, vegetable matter, and sulfur granules containing Actinomycetes organisms. The patient responded to bronchoscopic removal of the foreign body and penicillin therapy. To our knowledge, the association of actinomycotic infection with an aspirated endobronchial foreign body has not previously been reported.

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Actinomycosis causes a chronic suppurrative infection most commonly involving the cervicofacial region, thorax, and abdomen. The causative organisms, Actinomycetes, are slow-growing, filamentous, Gram-positive bacteria that are endogenous oral saprophytes. They dwell in carious teeth, dental plaque, and gingival and tonsillar crypts. Thoracic infection results from aspiration of infected oral contents, and may involve the lungs, pleura, mediastinum, or chest wall.

The infection typically spreads without regard to anatomic barriers, but involvement of major bronchi is rare. Primary endobronchial actinomycosis is exceptionally uncommon. We report the previously undocumented association of primary endobronchial actinomycosis with an aspirated endobronchial foreign body.

**CASE REPORT**

In June 1990, a 66-year-old man with a four-year history of non-insulin-dependent diabetes mellitus suffered an episode of choking while eating chicken. He developed a nonproductive cough that resolved spontaneously after two weeks. A week later, he underwent extraction of two carious lower teeth. Two years earlier, all of the patient's upper teeth were removed because of diffuse disease. The following day, the cough returned. His physician prescribed oral antibiotics for pneumonia. The cough persisted, and one week later, he noted fever that subsided coincident with one intramuscular injection of penicillin. Despite two additional courses of oral antibiotics, the cough continued. After two months of persistent symptoms and roentgenographic abnormalities, the patient was seen at the Mount Sinai Medical Center, New York.

On further questioning, he denied any history of alcohol abuse, seizure disorder, or episodes of loss of consciousness. Physical examination revealed a chronically ill man appearing older than his

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