measured pulmonary artery systolic pressure, which ranged from 52 to 92 mm Hg (mean, 74 ± 13 mm Hg) (Fig 2A). The pulmonary artery pressures calculated using the regression equation as described previously ranged from 52 to 91 mm Hg (mean, 72 ± 14 mm Hg). The pressures obtained by Doppler ultrasound were compared with the pulmonary artery pressure measured by Swan-Ganz catheterization and showed an excellent correlation (r = 0.98; standard error of the estimate = 2.8 mm Hg) (Fig 2B).

Nifedipine produced the most significant symptomatic and hemodynamic response, resulting in a decrease in pulmonary artery pressure to 52/26 mm Hg. The patient was discharged on oral therapy with nifedipine (30 mg every six hours), but because of marked edema of the ankles, the patient refused to continue the medication. A repeat Doppler examination one month after discharge from the hospital revealed a pressure gradient of 74 mm Hg between the right ventricle and the right atrium and a calculated pulmonary artery systolic pressure of 91 mm Hg, indicating a return to levels before treatment.

DISCUSSION

Numerous studies have been conducted to assess the response to various therapies in the initial and long-term management of primary pulmonary hypertension; however, until now, invasive monitoring has been necessary for serial evaluation.1,3 We have described a patient with primary pulmonary hypertension in whom an accurate diagnosis of pulmonary hypertension was initially made by using continuous-wave Doppler echocardiography. More important was our observation that the Doppler technique was sufficiently sensitive to detect even small changes in pulmonary artery systolic pressure during serial drug testing.

We believe that continuous-wave Doppler echocardiography represents a significant advance in the diagnosis and management of patients with primary pulmonary hypertension. This technique is not a substitute for initial Swan-Ganz catheterization; however, because it can accurately detect changes in pulmonary artery pressure, it may well alleviate the need for repeat invasive procedures. The test is easily performed at the bedside or in the office, causes the already ill patient no discomfort, and can be repeated serially as often as necessary, allowing closer monitoring of the patient on therapy.

REFERENCES


Farmer's Lung Presenting as Respiratory Failure and Homogeneous Consolidation*

Marc Chassé, M.D.; Gilles Blanchette, M.D., F.C.C.P.; Jacques Malo, M.D., F.C.C.P.; and Jean-Luc Malo, M.D.

A 40-year-old woman who worked on a farm and was exposed to moldy hay presented with acute respiratory failure requiring mechanical ventilation and homogeneous radiologic consolidation. Treatment with steroids produced rapid improvement. These features of presentation of hypersensitivity pneumonitis with acute respiratory failure and homogeneous consolidation are rarely encountered.

Acute hypersensitivity pneumonitis usually presents with normal chest radiographic findings or diffuse reticulonodular infiltrate which may cause functional impairment.1,3 It is unusual for this condition to present with respiratory failure and homogeneous consolidation as documented in the present report.

CASE REPORT

A 40-year-old non-smoking woman who worked on a farm and was exposed to moldy hay was transferred to our hospital because of pneumonia. She had been affected with dysnea and malaise for the previous six months. Two weeks before, she became progressively dyspeic and febrile. A chest radiograph showed a consolidation in the right upper lobe (Fig 1). The subject's clinical status deteriorated in spite of treatment with ampicillin and gentamycin.

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Figure 1. Posteroanterior chest radiograph taken at the time of acute illness demonstrating homogeneous consolidation of the right upper lobe.
plastic sheet. The subject had inhaled a whitish dust originating from this hay at least two hours a day while milking cows.

**DISCUSSION**

Several typical features which were highly suggestive of farmer’s lung were found in our subject. This woman, who worked on a farm and was exposed to moldy hay, reported shortness of breath. Her physical examination showed fever, tachypnea and inspiratory crackles. Leukocytosis and precipitins to two different extracts of *Micropolyspora faeni* were present. Lung biopsy revealed a picture which was typical of the condition.6,7 There was a rapid and remarkable improvement after using steroid therapy. The possibility of bacterial, fungal or viral infections was excluded. Confirmation of farmer’s lung through a bronchial provocation test with *M faeni* was considered. However, we were reluctant, for ethical reasons, to carry out these tests in a subject who had previously required mechanical ventilation.

Our subject developed two characteristics of hypersensitivity pneumonitis which are very rarely seen: respiratory failure and homogeneous radiologic consolidation. The functional abnormalities described in farmer’s lung rarely cause respiratory failure. Only one fatal case due to acute exposure to moldy hay in a 17-year-old youth who developed respiratory failure has been described.8 To our knowledge, only three cases of hypersensitivity pneumonitis with homogeneous consolidation in bird fanciers have been reported.8

We conclude that farmer’s lung can exceptionally present with acute respiratory failure requiring mechanical ventilation and homogeneous infiltrate of the lung.

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![Figure 2. Lung biopsy at the site of the right upper lobe consolidation showing diffuse inflammatory infiltrate and several noncaseating granulomas, some with giant cells, one of which is illustrated in the center of the figure (original magnification, ×50).](http://journal.publications.chestnet.org/pdfaccess.ashx?url=/data/journals/chest/21545/)