

症 例

Mitral Valve Replacement Complicated by Interstitial Pneumonia

Ryu Okutani*, Masaaki Iwaya*

Abstract

A 59-year-old male underwent mitral valve annuloplasty for mitral valve prolapse. The postoperative course was uneventful. However, three months after operation, he developed general malaise and dyspnea, and was readmitted to hospital. Echocardiography revealed mitral valve regurgitation (IV°), and chest x-ray showed an infiltrative shadow in the right lung. A diagnosis of interstitial pneumonia was confirmed based on an increase in serum KL-6 to 1,400 U/ml, and lung biopsy. Respiratory functions improved after about one month of mechanical ventilation and steroid pulse therapy, but 3 l/min of oxygen by nasal cannulae was still required. Chest x-ray revealed that the pneumonia had stabilized, although the serum KL-6 value remained high. Then, mitral valve replacement was performed. Extubation was done two days after surgery, and his respiration was stable thereafter. The serum KL-6 level decreased from 1,170 U/ml on the day of surgery to 541 U/ml 10 days after surgery.

Key words; interstitial pneumonia, serum KL-6, congestive hear failure, mitral valve replacement

Introduction

Interstitial pneumonia is a generic term for inflammatory pulmonary disease caused by inflammatory cellular infiltration into the alveolar septal interstitium^{1,2)}.

It can be classified by specifiable causes, such as pneumonia due to collagen disease, drug-induced and viral pneumonia, and idiopathic pneumonia in which the etiology is unknown^{3~10)}. KL-6 has recently been attracting attention as a serum marker that reflects the severity of pulmonary fibrosis in interstitial pneumonia^{11~16)}. Here, we report a case of interstitial pneumonia that occurred simultaneously with congestive heart failure accompanying acute aggravation of mitral valve regurgitation following mitral valve annuloplasty. Priority was initially given to providing general treatment, and once the respiratory and hemodynamic functions were stable, mitral valve replacement surgery was performed. As a result, a good surgical postoperative course was obtained.

Case Report

The patient was a 59-year-old male who was 169cm tall and weighed 55kg. His chief complaint was dyspnea. He had past histories of gout beginning 10 years previously and hypertension one year previously. His family history was unremarkable, but he had a smoking history of 40 cigarettes each day for 40 years. The patient underwent mitral annuloplasty for mitral valve prolapse and mitral regurgitation (IV°). Mitral valve annuloplasty was performed successfully, and the postoperative course was uneventful. The patient showed sufficient improvement to be discharged 40 days after surgery. However, three days after discharge he developed general malaise, and since his symptoms did not improve and dyspnea also developed, he was re-hospitalized for additional treatment 3 months after operation.

*Intensive Care Unit, Hyogo College of Medicine, Hyogo, Japan

Upon rehospitalization, the patient was lucid, with blood pressure of 110/60mmHg and a regular pulse rate of 60/min. Mild edema was seen in both lower extremities. The patient received oral diuretics and digitalis. A biochemical examination of the blood at the time of hospitalization revealed mild renal function disorder. Chest x-ray showed a cardiothoracic ratio of 52%, and vascular shadow enhancement was seen in both pulmonary hilar regions. Interlobar pleural effusion was evident in the right lung. Echocardiography showed no abnormalities in left ventricular wall motion, but left ventricle enlargement (LVDd, 67.9mm) and mitral valve regurgitation (IV°) were evident. Mitral valve repair surgery was thus indicated.

Subsequently, chest x-ray revealed pneumonia images and respiration also worsened. Despite administrations of diuretics and dopamine, the symptoms did not improve. The patient developed paroxysmal supraventricular tachycardia, with the heart rate suddenly jumping to about 180 beats/min. Blood pressure also decreased to 70/50mmHg, causing loss of consciousness, and the patient was admitted into the ICU. Since the renal function had also deteriorated, continuous filtration hemodialysis started. Hemodynamics were finally stabilized with catecholamine administration (5 μ g/kg per min of dopamine and dobutamine). The patient suffered nasal bleeding, and

respiration worsened together with aspiration, so endotracheal intubation was performed and artificial ventilation was required for four days.

A bronchoscopy was performed and a lung biopsy was conducted. Histopathological examination of the bronchoalveolar wall from the lung biopsy revealed Masson body formation on the alveolar wall centrally, and regeneration of large type II alveolar epithelium superiorly (Fig. 1). Alveolar wall hyperplasia, edema and lymphocytic infiltration were seen, and interstitial changes were also noted. There were no changes similar to those seen with pulmonary hypertension in alveolar microvessels, such as hyperplasia of the wall.

All tests for bacteria and viruses were negative and no fever was evident; hence, the patient was diagnosed of interstitial pneumonia. Steroid pulse therapy was conducted. This involved administration of methylpredonizolone at a dose of 1g/day followed by 500mg/day for two days. This improved the pneumonia based on x-ray images (Fig. 2) and pulmonary oxygenation. Cardiorespiratory functions also became relatively stable, and so mitral valve replacement surgery was undertaken.

During surgery, anesthesia was maintained with diazepam, fentanyl, and vecuronium. Under administration of 5 μ g/kg per min of dopamine and dobutamine, the arterial pressure was 110/60mmHg, pulmo-

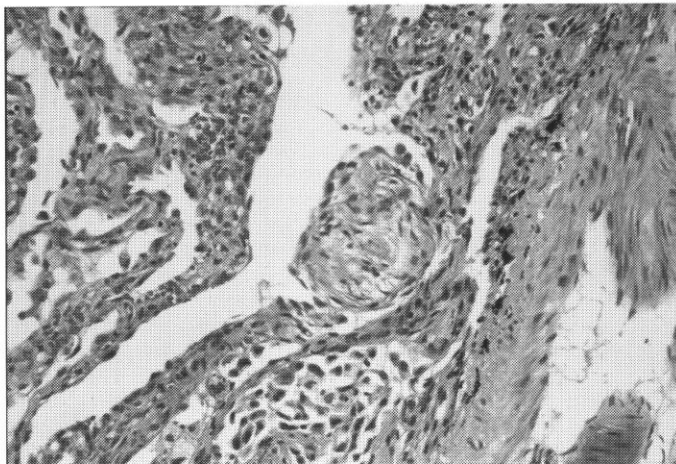


Figure 1 Histopathological findings from bronchopulmonary biopsy; histopathological image: Masson body formation and large type II alveolar epithelial regeneration are evident.

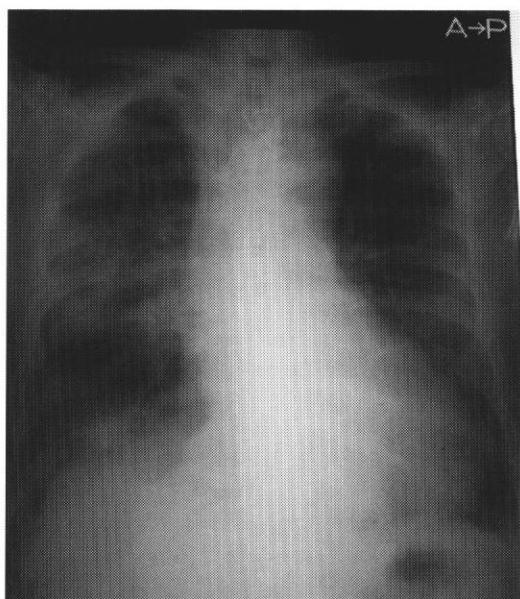


Figure 2 Chest X-ray before the second surgery

nary arterial pressure was 86/39mmHg, central venous pressure was 17mmHg, and cardiac index was 1.6 l/min per m².

Following withdrawal of the extracorporeal circulation (ECC), 10µg/kg per min of dopamine and dobutamine and 0.2g/kg per min of olprinone were required, and the pulmonary arterial pressure dropped to 32/22mmHg. One day after surgery, chest x-ray showed improvement in right lung infiltration, and extubation was done two days after surgery. Five days after surgery the patient was taken off catecholamines and only remained on oral administration of digoxin. On chest CT images 10 days after surgery, the pleural effusion and pneumonia shadows seen before surgery disappeared.

The serum KL-6 concentration was measured over time during the course of treatment. KL-6 increased from 644 U/ml before entry into the ICU to 1,690 U/ml at its peak. Despite treatment for about one month, it remained at a high level of 1,170 U/ml, at which point surgery was undertaken. This value started to fall rapidly from one day after surgery, and 10 days after surgery it fell to 541 U/ml.

Discussion

It is unclear whether the interstitial pneumonia was caused by pulmonary congestion and pulmonary hypertension accompanying mitral valve disease, since there have been no previous reports showing a relationship between pulmonary congestion and interstitial pneumonia. However, based on the postoperative course, respiration improved as cardiac function improved, the interstitial pneumonia shadow on x-ray disappeared, and the serum KL-6 concentration also fell, and hence it is inferred that there was some relationship between cardiac failure and interstitial pneumonia.

KL-6 is a high molecular weight glycoprotein categorized as an embryonic cell antigen (cluster 9)¹⁵. The utility of KL-6 as a serum marker for idiopathic interstitial pneumonia has been reported based on diagnostic and active evaluation, prognostic factors and treatment response assessment. For diagnosis of interstitial pneumonia, histopathological findings from lung tissue are the most reliable. But repeated sampling is difficult so KL-6 has attracted attention as a useful marker. In cases of interstitial pneumonia, KL-6 is highly expressed in type II alveolar epithelial cells, and is probably generated in such cells in high amounts. In particular, interstitial pneumonia is highly likely if the KL-6 value is 1,500 U/ml or higher^{16,17}. Furthermore, KL-6 also increases in cases of cardiac failure. It is therefore thought that these two factors are involved in the high preoperative serum KL-6 level in the present case. That is, it is highly likely that the pathology of the present case was an interstitial alteration in the air vesicles caused by pulmonary congestion and pulmonary hypertension accompanying mitral incompetence.

We were concerned about the timing of surgery. We should have promptly undertaken surgery once infection was ruled out, even in the presence of interstitial pneumonia complicating congestive heart failure and pulmonary congestion before surgery. By doing so, the conditions of the cardiac and pulmonary functions, including the pulmonary lesions, would probably have been improved.

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