3. The Pulmonary Gas Exchange of Chronic Respiratory Failure

CREF include tuberculosis and related conditions (43.3%), chronic obstructive pulmonary disease (43%), lung cancer (6%), and interstitial pneumonitis (5%). On average REF was observed in one third after 5 years and in half after 10 years from the onset of those underlying illness.

- (3) Acute exacerbation (AE) requiring hospitalization of the CREF were reported 2.5 times on average (1 – 10 times) during their observation period. AE was not more than 3 times for 77% of CREF.
- (4) Cumulative survival curve obtained with life table method indicated that survival rate was

62.5% at 3 years, 42.5% at 5 years and 34.5% at 7 years after the onset of REF. The survival rate was 60.7% after three times, and 44.8% after five times of A.E. The cause of death was attributable to respiratory disease in 77% of CREF.

- (5) The risk factors that carries poor prognosis for CREF include old age, right heart failure peptic ulcers and abnormal serum electrolytes. Factors that relates to favorable prognosis consist of those with COPD as underlying pathology, those who has longer history before the onset of REF, and those who has higher PaO_2 at the time of AE.

3. The Pulmonary Gas Exchange during the Acute Exacerbation of Chronic Respiratory Failure

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This study was conducted to investigate the impairment of gas exchange in the lungs during acute exacerbation of chronic respiratory failure. Respiratory failure was defined as the respiratory impairment in which arterial O_2 tension fell below 60 torr (or the patient shows the equivalent symptoms) while inhaling room air, as agreed by the research committee on respiratory failure of the Ministry of Health and Welfare of Japan. Chronic respiratory failure was defined as respiratory failure lasting for not less than a month. Acute exacerbation of chronic respiratory failure referred to those cases in which a further and rapid deterioration of chronic respiratory failure occurred to a condition which required more intensive treatment, preferably hospitalization.

SUBJECTS AND METHODS

Thirty-eight patients were studied in this investigation. All of them were inpatients who had been admitted to Keio University Hospital with acute exacerbation of chronic respiratory failure. Arterial blood gas examination was per-

formed on all of the subjects while inhaling room air, both during the stable and the acute exacerbation period of their chronic respiratory failure. The clinical diagnoses of the subjects were as follows: chronic pulmonary emphysema (CPE, 11 patients), interstitial pneumonitis and pulmonary fibrosis (FLD, 10 patients), post-thoracoplasty and/or callosity of the pleura and/or deformity of the thorax (thorax group, 10 patients) and others (7 patients). The total number of episodes of acute exacerbation experienced by the subjects was 78.

Right heart catheterization studies were performed on 5 of the 38 patients both during the stable period and during the acute exacerbation period. These studies enabled analysis of the distribution spectra of ventilation-perfusion ratios ($\dot{V}_A/\dot{Q}_C$) in the lungs and comparison of $\dot{V}_A/\dot{Q}_C$ uneven distributions during the acute exacerbation period and the stable period. In the analysis O_2 , CO_2 and N_2 were used as indicator gases, and no diffusion barriers for these indicator gases were assumed.

RESULTS

(1) Among the triggers which caused the episodes of acute exacerbations, the greatest number of cases, 54%, were related to respiratory infections. Right heart failure was associated with 25% of the cases. Some of the triggers of the acute exacerbation in the FLD group were thought to be the aggravation of the interstitial pneumonitis itself.

(2) During the stable period, the mean value for the arterial CO₂ tensions (P_{aCO_2}) in the CPE group was nearly equal to the upper limits of the normal P_{aCO_2} range, while in the thorax group the value was higher than the normal upper limits. The mean values for P_{aCO_2} increased further during the acute exacerbation period (Fig. 1). For both the CPE and the thorax groups the differences in the alveolar-arterial O₂ tension difference ($AaDO_2$) between the stable period and the acute exacerbation period were rather small, suggesting that the alveolar hypoventilation was an important factor in the impairment of the pulmonary gas exchange during the acute exacerbation in both these groups (Fig. 2). Thirty-seven per cent of the acute exacerbation episodes in the CPE group showed aggravation of the

alveolar hypoventilation which already existed in the stable period, and in 42% of the episodes in the CPE group there was development of alveolar hypoventilation which did not exist in the stable period. However, in 21% of these episodes in the CPE group there was no significant rise or fall in P_{aCO_2} during the acute exacerbation period, which indicated that there was no aggravation of the alveolar hypoventilation.

In the thorax group the alveolar hypoventilation during the acute exacerbation period followed the same pattern as in the CPE group.

The mean value for P_{aCO_2} in the FLD group in the stable period was very close to the lower limits of the normal P_{aCO_2} range and the mean value for P_{aCO_2} decreased further during the acute exacerbation period (Fig. 1). In this group the mean value of the differences in $AaDO_2$ between the stable period and the acute exacerbation period was rather large. This suggested that the mechanism which increased $AaDO_2$ (the aggravation of the $\dot{V}_A/\dot{Q}_C$ inequality and/or the diffusion impairment and/or the increase in shunted pulmonary blood flow) played an important role in the impairment of the pulmonary gas exchange during the acute exacerbation in the FLD group (Fig. 2).

Fig. 1. The arterial O₂ and CO₂ tensions in the thorax, CPE and FLD group during the stable and the acute exacerbation period. The arrows indicate the direction of the change of the blood gas measurement during the acute exacerbation periods. The thorax group: post-thoracoplasty and/or callosity of the pleura and/or deformity of the thorax; CPE: chronic pulmonary emphysema; FLD: interstitial pneumonitis and pulmonary fibrosis.

Fig. 2. The difference in arterial O₂ tensions (ΔP_{aO_2}), arterial CO₂ tensions (ΔP_{aCO_2}) and alveolar-arterial O₂ tension differences ($\Delta AaDO_2$) between the stable and the acute exacerbation period.

3. The Pulmonary Gas Exchange of Chronic Respiratory Failure

Fig. 3. Distribution patterns of the pulmonary blood flow and the ventilation against $\dot{V}_A/\dot{Q}_C$ on the logarithmic scale. These were obtained from a patient with chronic pulmonary emphysema. The results on the left and the middle panels were obtained during his stable status, at an interval of 2 years. The one on the right panel shows the result during his acute exacerbation period.

Thirty-five per cent of the acute exacerbation episodes in the FLD group showed aggravation of the hypocapnia which already existed in the stable period, and in 18% of the episodes in the FLD group there was development of hypocapnia which did not exist in the stable period. However, 12% of the episodes in the FLD group showed a significant rise in P_{aCO_2} during the acute exacerbation period, which indicated that alveolar hypoventilation might be involved in the impairment of pulmonary gas exchange.

(3) The impairment of pulmonary gas exchange did not always follow the same pattern in individual patients during repeated episodes of acute exacerbations.

The pulmonary gas exchange did not necessarily deteriorate more with further episodes of the acute exacerbations.

(4) There was no significant difference in arterial gas measurements taken during the acute exacerbation periods of patients who recovered and those who died. This indicated that the prognosis of the patients during acute exacerbation might not depend solely upon the impairment of the pulmonary gas exchange, but that

many other factors and their interrelations might also be involved.

(5) During acute exacerbation of chronic respiratory failure, a decrease in the mixed venous O_2 tension was found in all the cases examined and either a broadening of the distribution spectra of $\dot{V}_A/\dot{Q}_C$ or the occurrence of a new mode of pulmonary blood flow at low $\dot{V}_A/\dot{Q}_C$ units was observed (Fig. 3).

CONCLUSION

- 1) These studies have shown a difference in impairment of the pulmonary gas exchange during the acute exacerbation period among the CPE, FLD and thorax groups of chronic respiratory failure.
- 2) The impairment of pulmonary gas exchange did not always follow the same pattern in individual patients during repeated episodes of acute exacerbations.
- 3) During the acute exacerbations either a broadening of the distribution spectra of $\dot{V}_A/\dot{Q}_C$ or the occurrence of a new mode of pulmonary blood flow at low $\dot{V}_A/\dot{Q}_C$ units was observed.