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Inflammation of the Bronchial Tree

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Abstract

Ventilatory disturbances in CLE cannot be accounted for only by decrease of internal surface area. Indeed, in the majority of the cases studied the intact parenchyma accounted for 50 to 77% of the volume of the lungs and the internal alveolar surface area was only slightly reduced. These results, as previously stressed by Dunnill, are the reverse of the findings in severe PLE cases in which there is frequently minimal respiratory failure and right ventricular hypertrophy, although emphysema may destroy as much as 80% of the lung parenchyma, thus seriously reducing the internal ventilatory surface area.

Keywords: Ventrilator surface; Lung parenchyma; Bronchiolar narrowing; Respiratory failure; Inflammatory stenosis; Emphysema

Introduction

Staub and Gomez emphasized that the ventilatory disturbances in CLE were mostly due to the enlarged centriacinar spaces, which were situated in a strategic position between the conductive zone and the respiratory exchange area, and slowed down gas diffusion. As a result, Dunnill pointed out that the number and the distribution of these abnormal air spaces were a more reliable guide than their size and the total lung volume involved. However, the predominant location of the centri-lobular foci in the upper half of the lung demonstrated here as well as by Snider et al. and Thurlbeck, suggests that this theory cannot account for all the facts. Indeed, more than half of the lung parenchyma and particularly the lower halves of the lungs, which are the best perfused and ventilated, generally show much less centri-lobular emphysema. This suggests that other mechanisms are responsible; especially increase of terminal airways resistance. Leopold and Gough noticed inflammatory narrowing's in 12% of the respiratory bronchioles supplying CLE spaces, and narrowing of membranous bronchioles was not reported by them. The present morphometric study of the membranous bronchioles showed that bronchiolar narrowing's were scattered randomly inside the lung, including zones without emphysema. Moreover, the degree of these bronchiolar narrowing's seemed related to the severity of chronic respiratory failure. This inflammatory stenosis situated at the end of the conductive air passages could play an important part in ventilatory disturbances, as discussed in a previous paper, and were demonstrated in recent physiological studies by Hogg [1]. Right ventricular hypertrophy was noted in 55% of the 75 cases studied by Leopold and Gough, and the majority of authors have noticed the frequency of RVH in CLE even in cases with moderate emphysema and sometimes at an early stage. Nevertheless, the cause of the PAH or RVH is often uncertain or controversial. Permanent structural changes in the pulmonary arterial system are a matter of debate, and even if pulmonary arterial destruction occurs, it is of minor importance as a cause of chronic pulmonale in CLE, because in some cases RVH develops with only moderate parenchymal destruction. Compression of the pulmonary arterioles by the centri-lobular air distended spaces, which was suggested by Dunnill as a cause of pulmonary hypertension, was not found in this study. Heath showed that the small pulmonary arterial vessels often presented characteristic histological features in emphysema when there was associated right ventricular hypertrophy. These changes included development of longitudinal muscle in the intima of pulmonary arterioles, the development of medial circular muscle in the pulmonary arterioles, and a lack of hypertrophy of the medial muscle in the large muscular pulmonary arteries. This triad of structural changes might account for the increased pulmonary vascular resistances in CLE [2].

Discussion

Such vascular changes, however, are not only characteristic of emphysema but are found in all conditions of chronic hypoxia. The potentially reversible nature of these arterial changes is probably related to the reversibility of pulmonary arterial hypertension following relief of chronic hypoxia. In six cases of the present series there were no major structural changes in the muscular pulmonary arteries and pulmonary arterioles [3]. The pulmonary arterial vessels, however, were not subjected to quantitative studies, as proposed by Arias-Stella and Saldafia, Naeye or Hicken. Thus detection of mural changes in distal pre-capillary arterioles, similar to those observed in healthy subjects living at a high altitude, in kypho-scoliosis or in the Pickwickian syndrome may have been missed. Only in two cases were permanent and irreversible vascular changes found to explain the extent of RVH, and these consisted of intimal fibrous thickening causing narrowing of the arterioles and, to a lesser extent, of the muscular arteries. These changes most probably resulted from organized scattered thrombo-emboli. Thus pulmonary thrombo-emboli, which are frequently found in chronic broncho-pulmonary disease, may help to increase the pulmonary hypertension in some cases. In our cases, RVH and, in consequence, PAH appeared to be related to the quantitative structural damage to the lung parenchyma and bronchioles. Of these changes, bronchiolar stenosis played the more important part and its importance, seven when minimal emphysema is present, has been stressed previously. It is questionable; however, whether the high pressures generated beyond bronchiolar narrowing's during coughing could disrupt respiratory bronchioles, as suggested by McLean [4]. This would not account for the localization of centriacinar holes in the lungs. Pulmonary hypertension appears to result from functional factors, especially hypoxia, caused by structural changes in the airways. Thus,

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a new structural model can be constructed to account for ventilatory obstacles in CLE. In the upper half of both lungs there are two obstacles 'in series', first, bronchiolar narrowing in the conduction zone and, second, centriacinar dilated air spaces in the intermediate zone. In the lower halves of the lungs there is mainly bronchiolar narrowing before the zone of diffusion. Thus, these two obstacles interfere with the transfer of respiratory gases in a zone where good gas conduction and diffusion are most needed. Because the centriacinar branches of the pulmonary artery generally remain intact, there is good vascular perfusion but little or no ventilation at the level of the peripheral alveolar exchange zone. Therefore, in CLE a significant ventilation perfusion abnormality occurs throughout the lung, even though parenchymal destruction is moderate and localized to its upper portion [5]. Pulmonary arterial hypertension and the resulting right ventricular hypertrophy is a sequel to muscularization of the pulmonary arterioles caused by hypoxia, but in some cases these changes in the pulmonary vessels are increased by wide-spread organized thrombotic emboli. The mucous gland hyperplasia was demonstrated by two different quantitative methods which gave similar results. Whereas clinical chronic bronchitis was found in all cases, mucous gland hyperplasia was found only in seven. No other important changes or localized bronchiectasis were detectable. In centri-lobular emphysema, parenchymal destruction is situated at the level of the respiratory bronchioles, producing initially enlarged centriacinar air spaces but leaving more or less intact alveoli distal to these spaces [6]. The centriacinar holes may join to form larger centri-lobular spaces. This type of emphysema is usually associated with chronic bronchitis and is thought to result from respiratory bronchiolitis. During its course, and often at an early stage, it usually results in severe respiratory failure with right ventricular hypertrophy. The ventilatory disturbances produced by centri-lobular emphysema have been shown by the use of a model to be almost exclusively caused by an increased diffusion time of gas molecules through distended centriacinar or centri-lobular spaces before they reach the respiratory exchange area of the lung. However, such a model hardly accounts for the severity of most of the changes observed in cases of centri-lobular emphysema. Usually only the upper zones of the lung and less than 40% of the parenchymal volume show centriacinar spaces, and it is uncertain whether, in addition to the centriacinar spaces, some other obstacle to alveolar ventilation may be present throughout the lung. Such an obstacle might be caused by bronchiolar narrowing's which were described by McLean [7]. Previously we have shown, using a quantitative method, that widespread bronchiolar stenosis were present in chronic obstructive broncho-pulmonary disease with or without emphysema. Recently it has been emphasized that in chronic obstructive pulmonary disease the obstruction to airflow is situated in small bronchi of less than 2 mm. diameter [8]. The present study, employing morphometric methods, was designed to investigate the quantitative relationship between the alveolar and bronchiolar damage in centri-lobular emphysema, and the severity of the resulting chronic pulmonary hypertension and right ventricular hypertrophy [9]. The importance of permanent structural changes in the pulmonary arteries as a cause of the pulmonary hypertension was also investigated. Measurements of lung parenchyma, membranous bronchioles, and bronchial mucous gland hyperplasia were made on lungs from eight cases of pure centri-lobular emphysema and on five normal lungs. The lungs were fixed in formalin and inflated under partial vacuum at a standard trans-pulmonary pressure of +30 cm. H20. The results obtained from the upper halves and the lower halves of the lungs were compared. The circulatory effects of the disease were measured by weighing the heart ventricles, by studying the small pulmonary arteries in microscopical sections, and by post-mortem arteriography [10]. Whereas the parenchymal and internal surface areas destroyed by the emphysematous spaces were relatively moderate and localized, right ventricular hypertrophy was noted in most of the cases. In these cases bronchiolar stenosis were found scattered throughout the whole lung and there was a reduction in the number of these bronchioles, mainly in the upper halves of the lungs.

Conclusion

In centri-lobular emphysema ventilatory disturbances were caused not only by the centriacinar dilated spaces delaying gas diffusion, but also by scattered bronchiolar stenosis situated at the termination of the conducting air passages. The stenosis seemed the more important cause. It was shown statistically that chronic arterial pulmonary hypertension and right ventricular hypertrophy were mainly the result of functional disturbances, especially hypoxia and abnormalities of VA/Q produced by the two structural changes situated at the end of the small airways.

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Conflict of Interest

None

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