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Editorial

The Morphology of Pulmonary Involvement in COVID-19 Infection Stimulates and Requires a Differentiating Terminology in Inflammatory Lung Diseases

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Abstract

The paper shows using the example of Covid-19-infection, that it is necessary to define pulmonary infections and alterations on the basis of morphology. Using the term pneumonia only is misleading and may be harmful for patients with lung parenchyma infections.

Keywords: *Pneumonia; Covid-19; Lung Parenchyma Infection; Alveolitis*

Although COVID-19 is a general infection [1,2], the lung changes essentially characterize the clinical picture. So far, they have been considered mainly from a therapeutic point of view, as they are decisive for the prognosis. Indeed, increasing experience has contributed to a better understanding of the course of the disease and better therapeutic results.

In the attempt to gain a deeper understanding of the processes in the lung tissue, the morphological situation in the periphery of the lung tissue is essential, as this directly causes the functional disorders of the lung. In contrast to the extensive therapeutic literature on COVID-19, only few data are available on the relationship of the morphological findings in the lung tissue concerning the respiratory insufficiency that dominates the events in the severe stage of the disease. The following text addresses this problem and hypothetized that a more precise terminology of inflammatory peripheral lung changes would create better results in therapy of those diseases. Unfortunately, there is no general accepted terminology for inflammatory lung parenchyma alterations and diseases.

The morphology of the peripheral lung tissue in COVID-19 disease shows significant tissue alteration in the alveolar walls with cellular infiltration by inflammatory cells, fluid accumulation and microthrombi in the capillaries. These findings are described as diffuse alveolar damage (DAD) [1,3]. A striking feature is a marked increase in type II pneumocytes, which is not seen in this form in other inflammatory lung parenchymal diseases [4]. Viruses are detectable in both types of alveolar cells, but prefer the type II cells [6]. This suggests a relationship to the surfactant system of the lung, since the type II cells are the production site of surfactant substances. Initial molecular biological findings and pathophysiological considerations indeed point to impaired surfactant production in COVID-19 infection [5,6]. A disturbance of the surfactant system has been described for classic ARDS as a consequence of sepsis, etc [7]. More recent data show rela-

tions between COVID infection of the lung and a disturbance of the surfactant system [5,8]. Therapeutic approaches have been attempted [9]. A possible direct antiviral function of pulmonary surfactant substances may also be important in the context of infection. Considering alveolar cell function and morphology COVID-19 in the lung is clearly different from other alterations of lung parenchyma.

It is clear that the morphology of the COVID-19 lung, which appears as a total destruction of the affected tissue, must result in a significant reduction in local compliance, the microthrombi and inflammatory processes in the alveolar vessels, which have also been seen several times, are likely to increase rather than reduce this functional disturbance, since they do not permit or at least make more difficult the adaptation of perfusion to ventilation (hypoxemic pulmonary vasoconstriction) as a result of the inflammatory processes. The designation of these changes as DAD - diffuse alveolar damage - gives a vivid picture of the result of the disease process, but describes only a global view of the actual situation in the tissue. The main clinical symptom of respiratory insufficiency resulting from this morphology is hypoxemia.

Looking at the tissue changes, distribution disorders (V/Q disorder), locally and confluently in the course of the disease, are the most likely cause for this hypoxemia.

The radiological findings show a very uneven distribution of foci in the lung tissue.

Only few data are available on the interaction of local V/Q ratios in individual tissue areas [10]. They show complicated V/Q ratios, different in different diseased and non-diseased tissue areas. In cases with a high shunt volume, it is presumably also due to the disturbed or switched-off vascular control [10,11]. The cause of the hypoxemia is probably in the severely affected lung areas veno-arterial shunt on the one hand, and on the other hand a V/Q disturbance in the less affected areas with relatively good compliance due to hyperperfusion [12]. This is also supported by the experience of success with NIV. Structural destruction additionally affects fluid content and transport, which requires negative interstitial pressure under regular conditions, where normal surfactant function is playing an important role [13]. This function requires a normal lung structure, which is not present in the lungs affected with COVID-19. It is important that the infiltrations develop locally and then producing so-called milky glass-like images in X-ray. The fact that breathing can be improved by changing the patients position brings fluid shifts in the lung tissue into focus, in addition to a redistribution of blood flow [10,14]. The lung changes in COVID-19 are not very different to usual ARDS, but show special features that can be explained by the different morphology [10,12].

The morphological correlate of Covid-19 infection in the lung is a local but multilocular profound inflammatory tissue change with cellular infiltration of the alveolar walls, a disturbance of the lung mechanics and the interstitial and intraalveolar fluid dynamics and a possible additional damage to the pulmonary surfactant - thus the picture of alveolitis. This does not differ in principle but morphologically, depending on the cause, from other forms of alveolitis as seen in other viral diseases [15]. However, the histological findings in COVID 19 in no way resemble those of bacterial pneumonia, which is defined by an intraalveolar infiltrate with largely preserved alveolar walls [15]. The use of the diagnosis pneumonia is not justified in Covid-19 unless additional secondary bacterial infections are involved.

For non-pneumonic lung involvement, the term pneumonitis has been repeatedly proposed but has not systematically gained acceptance. The term pneumonitis as well as the term - interstitial pneumonia, which would also be applicable to Covid-19 changes, should be not used because there is practically no interstitial space in the peripheral lung and the term pneumonitis is not standardized internationally. The term pneumonia should only be applied to cases caused by bacteria. The different morphology of the lung changes depending on their etiology and pathogenesis requires a differentiated terminology. Without doubt a better understanding of the disease processes leads also to better therapeutic concepts. The term alveolitis points directly to the location of the disease process. COVID-19 can be seen as a particular example of an infectious disease of the lung. Other inflammatory changes with different morphology may occur in the future. It is therefore important to develop concepts of terminology of in etiology and pathogenesis very different inflammatory lung tissue diseases. A precise terminology is the basis of understanding diseases, as well as basis of an individual therapy.

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