

Pulmonology And Immunology; A Brief Overview

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Abstract

Pulmonary diseases are very common worldwide and have high mortality and morbidity rates. When we look at the pathogenetic processes of these diseases, it is seen that the natural and adaptive immune response plays an important role. As in many diseases, immune modulatory therapy is the current treatment approach in pulmonary diseases. In our article, we aimed to take a quick look at the immune system in common pulmonary diseases.

Keywords: chronic obstructive pulmonary disease (COPD), asthma, lung diseases

Introduction

Pulmonary diseases such as chronic obstructive pulmonary disease (COPD), and asthma, are very common lung diseases all over the world but unfortunately, we don't have any curative treatment options for these diseases. The treatment that we use, targets symptom control. Lung cancer is the second most common cancer in the world. Lung cancer ranks first among cancer-related deaths [1]. Treatment of diseases is possible with the correct understanding of pathophysiological processes. When we look at the pathophysiological processes of lung disease, immune system elements also stand out. In our article, we aim to discuss the role of the immune system in common lung diseases.

COPD is the third most common cause of death in the world. It progresses with symptoms such as dyspnea, cough, and sputum that increase with exertion, especially as a result of damage to the airways caused by cigarette smoke and air pollution. COPD; is a heterogeneous and often progressive disease in which airway inflammation such as bronchitis and bronchiolitis or alveolar damage resulting in emphysema is seen [2]. Respiratory tract infections are more common in these patients than in the normal population. Infections play an important role in disease pathogenesis and progression. However, the mechanisms involved have not been fully elucidated. COPD pathogens

such as Haemophilus influenza, Moraxella catarrhalis, and Streptococcus pneumonia are strongly associated with acute exacerbations of COPD. It is thought that the colonization of these pathogens in stable COPD patients causes the continuity of the harmful immune response in the pathogenesis of COPD [3]. Bronchial biopsies show an increased number of macrophages, neutrophils, T and B lymphocytes, and dendritic cells in the small airways of stable COPD patients [4]. The dominant cell type differs according to the stage of the disease. Especially neutrophil and B lymphocyte counts are significantly increased in advanced COPD patients [5]. The first immune response of the lung to cigarette smoke and harmful gases is mediated by alveolar epithelial cells, dendritic cells, and alveolar macrophages. The local immune response is enhanced by a variety of secreted cytokines, chemokines, and proteases. It has been shown that NF- κ B activation may be responsible for the increased expression of proinflammatory cytokines such as IL-1, IL-6, IL-8, MCP-1, TNF- α , and ICAM-1 in COPD [6]. In addition, some studies have shown high expression of Th17-related cytokines in the lower respiratory tract of moderate and severe COPD patients (IL-17, IL-22) and it has been concluded that this plays a role in the maintenance of neutrophils [7].

Asthma is a very common chronic pulmonary disease with variable airway limitation and exacerbated by allergens, exercise, irritant gases, or air change [8]. The relationship between asthma and the immune system has been investigated for a long time. However, the relationship between the pathogenesis of asthma and the immune system has not been fully clarified. Although it is known that Th-2 cells and cytokines such as IL-4, IL-5, IL-13, and Ig-E are closely related to asthma, studies have shown that neutrophils are dominant in a group of asthmatic patients. This group, which is called the group with low type-2 inflammation, is closely related to obesity and is unresponsive to steroid treatment [9]. In addition, it has been understood in some studies that the lung epithelium is not just a set of cells that act as a barrier but also acts as an orchestra conductor that provides the formation of the immune response against external stimuli. It has been observed that the expression of pattern-recognizing receptors such as TLR, NOD-like receptors, and RIG-like receptors from the lung epithelium play a role in shaping the immune response [9-10].

Immune treatment options for asthma patients have been applicable in the last 10 years. Anti-Ig-E (omalizumab), anti-IL-5 (mepolizumab), IL-5 receptor antagonist (benralizumab), and IL-4 receptor antagonist (dupilumab) are effective treatment options in selected patients who cannot be controlled despite high-dose inhaled or systemic steroid treatments [8].

The relationship between the immune system and cancer has been a subject that has been widely discussed in the last hundred years. The possibility of treatment of malignant diseases by immune modulation is very valuable for the scientific world. Two researchers, Burnet and Thomas, put forward the view that the development of cancer is prevented by the adaptive immune response in people with a healthy immune system. In the first studies, this thesis was supported at a low rate, but the place of IFN-gamma in the immunological rejection of planted cancer cells was demonstrated by conducting animal studies in which genetic-based immunodeficiency was created. In addition, the role of T cells, B cells, and NK cells in the prevention of cancer development has been clarified. Today, it is known that the protective role of the immune system in the development of cancer occurs in 3 ways. The first of these is to prevent the development of virus-based cancers (such as a cervical cancer-HPV relationship) by protecting against viral infections. The second is to eliminate pathogens and prevent

mediators that will emerge through the inflammatory process from playing a role in tumor development. The third way, for which we still do not have enough data and research are intensified, is that the innate and adaptive immune response recognizes tumor antigens and eliminates cancer cells [11]. These ways, which try to explain the relationship between the immune system and malignant diseases, form the basis for the idea of a 'cancer vaccine', as well as new treatment options for malignant diseases besides surgery, chemotherapy, and radiotherapy. Many researchers look at lung cancer from this perspective. Similar to other cancer types, it has been shown that local immune responses are decreased in lung cancer. It is known that especially antigen-presenting cells (APC and T cells) are decreased [12].

Conclusion

It is clear that lung diseases are an important cause of mortality and morbidity worldwide. Like many chronic diseases, it is closely related to quality of life and workforce. The development of curative treatments is possible with a good knowledge of the pathogenetic processes of diseases. The role of the immune system in the pathogenetic processes of pulmonary diseases is substantial. The collaboration of immunology and pulmonology will open new horizons in many stages from diagnosis to treatment.

Conflict Of Interest

The authors declare no conflict of interest.

References

1. <https://www.who.int/news-room/fact-sheets/detail/cancer>
2. GOLD-2023-ver-1.1-2Dec2022_WMV.pdf.
3. Leung, J. M., Tiew, P. Y., Mac Aogáin, M., Budden, K. F., Yong, V. F. L., Thomas, S. S., ... & Chotirmall, S. H. (2017). The role of acute and chronic respiratory colonization and infections in the pathogenesis of COPD. *Respirology*, 22(4), 634-650.
4. Barnes, P. J. (2014). Cellular and molecular mechanisms of chronic obstructive pulmonary disease. *Clinics in chest medicine*, 35(1), 71-86.
5. Hogg, J. C., & Timens, W. (2009). The pathology of chronic obstructive pulmonary disease. *Annual Review of Pathology: Mechanisms of Disease*, 4, 435-459.
6. Di Stefano, A., Caramori, G., Oates, T., Capelli, A., Lusuardi, M., Gnemmi, I., ... & Adcock, I. M.

- (2002). Increased expression of nuclear factor- κ B in bronchial biopsies from smokers and patients with COPD. *European Respiratory Journal*, 20(3), 556-563.
7. Caramori, G., Casolari, P., Barczyk, A., Durham, A. L., Di Stefano, A., & Adcock, I. (2016, July). COPD immunopathology. In *Seminars in immunopathology* (Vol. 38, pp. 497-515). Springer Berlin Heidelberg.
 8. <https://ginasthma.org/wp-content/uploads/2022/07/GINA-Main-Report-2022-FINAL-22-07-01-WMS.pdf>
 9. Hammad, H., & Lambrecht, B. N. (2021). The basic immunology of asthma. *Cell*, 184(6), 1469-1485.
 10. Willart, M. A., Deswarte, K., Pouliot, P., Braun, H., Beyaert, R., Lambrecht, B. N., & Hammad, H. (2012). Interleukin-1 α controls allergic sensitization to inhaled house dust mite via the epithelial release of GM-CSF and IL-33. *Journal of Experimental Medicine*, 209(8), 1505-1517.
 11. Schreiber, R. D., Old, L. J., & Smyth, M. J. (2011). Cancer immunoediting: integrating immunity's roles in cancer suppression and promotion. *Science*, 331(6024), 1565-1570.
 12. Sharma, S., Zhu, L., Srivastava, M. K., Harris-White, M., Huang, M., Lee, J. M., ... & Dubinett, S. (2013). CCL21 chemokine therapy for lung cancer. *International trends in immunity*, 1(1), 10.
 13. Johnson SK, Kerr KM, Chapman AD, Kennedy MM, King G, Cockburn JS, Jeffrey RR.(2000) Immune cell infiltrates and prognosis in primary carcinoma of the lung. *Lung Cancer*. Jan;27(1):27-35.