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A Short Note on Severe Refractory Eosinophilic Asthma

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Editorial

Asthma is a diverse disease in which adequate asthma control cannot be achieved in a significant proportion of patients despite currently available treatment options. Asthma in this subgroup is known as "severe refractory." Significant progress has been made in recent years toward a more precise definition of severe refractory asthma. A methodical approach to evaluating asthma patients has been proposed. Further classification into distinct phenotypes is being carried out in order to target the right treatment to the right patient. Furthermore, new therapeutic targeted treatment options are being developed to provide potential new targets for improving disease state, symptoms, and quality of life [1].

Severe eosinophilic asthma is a complicated disease, and much research has been conducted to fully comprehend its mechanisms. Bronchial remodelling and loss of lung function are important features of asthma, but their key aspects remain unknown, particularly the impact of biological drugs on them. Interleukin-5 (IL-5) is a key cytokine involved in the pathophysiology of eosinophilic asthma. It collaborates with other type 2 cytokines and chemokines in the development, transmigration, and persistence of eosinophils into airways, including eotaxin-2 and 3, thymic stromal lymphopoietin (TSLP), IL-33, IL-4, and IL-13 Several monoclonal antibodies (mepolizumab, reslizumab) have been developed to target this cytokine or its receptor (benralizumab) [2].

The majority of asthma patients can be effectively treated with the medications that are currently available. However, a significant proportion of patients labelled as having "severe refractory asthma" continue to pose difficulties for the treating clinician. Severe refractory asthma refers to a group of subtypes of asthma that do not respond to current standard therapy, which includes high doses of inhaled glucocorticoids combined with long-acting 2-agonists (LABA). The management of severe asthma is associated with an excessive and disproportional use of healthcare resources. Furthermore, there is a significant unmet clinical need [3]. As a result, a great deal of research is currently being conducted on topics such as assessment and evaluation, subphenotyping, and novel treatment modalities for severe asthma.

Current guidelines (Global Initiative for Asthma, National Asthma Education and Prevention Programme, and British Thoracic Society) recommend that patients with severe asthma be treated with high-dose inhaled or oral glucocorticoids in combination with LABAs and/or additional controller medications. Several biological pathways have been implicated in the pathogenesis of asthma, and these pathways do not always overlap with the clinical phenotypes described above. As a result, phenotyping at multiple levels is critical for identifying responders to targeted therapy. Several treatment targets have been identified, and several drugs are now being tested [4].

The first step in management is to identify the true "severe refractory asthma" patient. The patient's phenotype should be as accurate as possible in the second step. Until now, only clinical phenotyping has been used to guide treatment in clinical practise. Unbiased cluster analysis has already revealed three to four severe asthma subphenotypes. Integrated high-dimensional data will almost certainly lead to more accurate phenotyping in the near future. This will undoubtedly improve our understanding of the patho-immunobiology of the various asthma phenotypes and will assist clinicians in better predicting the natural history and prognosis of an individual asthma patient [5]. The most important outcome of this systems medicine approach, however, will be the development of new and improved treatment targets/strategies for this complex group of patients.

Conflict of Interest

None.

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