Asthma is one of the most common noninfectious chronic obstructive diseases of the respiratory tract. It affects approximately 5% to 7% of the population worldwide, including Poland.1 The basic objective in asthma treatment is to control disease symptoms by appropriate pharmacotherapy, as well as to reduce the risk of severe exacerbations.2 However, in approximately 5% to 10% of patients, these targets are not achieved despite administration of standard medications at the highest therapeutic doses. In such cases, the condition is termed “severe asthma,” which is defined by the World Health Organization as an uncontrolled disease associated with the risk of frequent, severe exacerbations (or death) and/or adverse side effects of the treatment and/or impairment of pulmonary function.3 The term severe asthma is also used to describe patients who, within the preceding year, required a large dose of an inhaled glucocorticoid together with an inhaled long-acting β2-agonist, or used oral glucocorticoid for at least 6 months, but the disease remained uncontrolled despite treatment.3

Severe asthma (ie, asthma resistant to treatment) is problematic both for the patient (as it is associated with poor quality of life, adverse effects of medications, frequent and unexpected exacerbations and hospitalizations) and for the health care system. To improve the therapy of severe asthma, attempts have been made to identify asthma phenotypes and develop an individualized and phenotype-directed treatment approach. The phenotypes include severe allergic asthma, which is challenging both for physicians and patients. In allergen-induced asthma, immunoglobulin E (IgE) and T helper cell 2-mediated eosinophilic inflammation play an important role.5 In early allergic reaction, exposure to allergens results in the release of proinflammatory mediators, which are responsible for contraction of the airway smooth muscle and increased mucus production, leading to the development of clinical symptoms, such as wheezing, shortness of breath, chest tightness, and cough. In late allergic response, bronchial infiltration of inflammatory cells is observed, which leads to tissue remodeling and irreversible respiratory obstruction.6,7

Omalizumab, the first antibody introduced to the market for a wide application in asthma therapy, binds free IgE in the circulation and prevents its attachment to the surface of inflammatory cells. The reduction of free IgE levels in serum results in a decrease in the levels of receptors for IgE on inflammatory cells, preventing them from responding to allergens.8,9 In clinical trials, omalizumab was shown to reduce the rates of asthma exacerbation and hospitalizations, reduce daily inhaled and oral glucocorticoid doses, and improve the quality of life. It was also well tolerated, with an adverse event profile similar to that of placebo.10,11 Omalizumab was approved for the treatment of severe allergic asthma by the Food and Drug Administration in the United States in 2003 and by the European Union in 2005. Also in Poland, since 2013, patients with severe allergic asthma who are aged 12 years or older and who have positive skin prick test results or in vitro reactivity to perennial aeroallergens, may be treated with this antibody within the national treatment program of the Polish National Health Fund.2,12

The inclusion and exclusion criteria for this program have been determined based on Appendix B.44 of the Minister of Health Official Journal: “Treatment of severe IgE-dependent allergic asthma with omalizumab.”13 The most important criteria in this program are the use of an oral glucocorticoid in the preceding 6 months and fulfillment of at least 3 of the following conditions: 1) significantly impaired quality of life due to asthma symptoms; 2) 3 or more severe exacerbations in the previous year, which required the use of or an increasing dose of systemic glucocorticoid; 3) hospitalization due to asthma exacerbation within the previous year; 4) a history of a life-threatening incident of an asthma attack; and 4) persistent

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The median IgE level was 203 IU/ml (range, 100–431 IU/ml). During treatment, we observed a reduction in oral glucocorticoid dose (a decrease in the number of exacerbations, and a decrease in peripheral eosinophilia, differences in the predicted value). The most common allergens, with confirmed relation to asthma symptoms, were house dust mites (n = 22; 71%), mold (n = 7; 23%) and cat dander (n = 12; 39%). The median IgE level was 203 IU/ml (range, 100–431 IU/ml). During treatment, we observed an improvement in the quality of life, a decrease in the number of exacerbations, and a reduction in oral glucocorticoid dose. Despite the trend for better lung function and decrease in peripheral eosinophilia, differences were not significant.

In conclusion, therapy with omalizumab for severe asthma is available in daily practice as an add-on treatment also in Poland. Currently, more than 90% of our treated patients are recruited from the city of Kraków and its vicinity. We believe that there are many more eligible patients in smaller cities and rural areas of Poland. With appropriate knowledge of the inclusion criteria, not only pulmonologists but also internists might refer patients to centers offering omalizumab therapy.

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**Conflict of interest**
The authors declare no conflict of interest.

**How to cite**

**REFERENCES**

**TABLE 1**
Clinical and laboratory characteristics of patients with asthma at baseline and after 104 weeks of omalizumab therapy

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Baseline</th>
<th>104 weeks of treatment</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Oral corticosteroid dose, mg, recalculated to methylprednisolone</td>
<td>8 (4–16)</td>
<td>0 (0–8)</td>
<td>0.0002</td>
</tr>
<tr>
<td>Asthma Control Questionnaire, points</td>
<td>3.1 (2.8–3.5)</td>
<td>2 (1.1–3)</td>
<td>0.0003</td>
</tr>
<tr>
<td>Asthma Quality of Life Questionnaire, points</td>
<td>3.2 (2.8–3.87)</td>
<td>5.1 (4.5–6.3)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Forced expiratory volume in 1 second, % of the predicted value</td>
<td>66 (55–83)</td>
<td>77 (71–84)</td>
<td>0.29</td>
</tr>
<tr>
<td>Exacerbations in the last year, n</td>
<td>4 (3–6)</td>
<td>0.5 (0–2)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Blood eosinophilia, number/µl</td>
<td>240 (120–400)</td>
<td>160 (90–250)</td>
<td>0.12</td>
</tr>
</tbody>
</table>

Data are presented as median (range). A P value of less than 0.05 was considered significant.

respiratory obstruction (forced expiratory volume in 1 second <60% of the predicted value or daily peak expiratory flow changes >30%).