“Breathe easier my child”

Kids & Asthma

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Asthma is an interplay between inflammation, structural airway changes and airway responsiveness. Airway inflammation is characterized

In this article:

1. How do I diagnose asthma in children?
2. What asthma medications are available?
3. What are the roles of leukotriene receptor antagonists and long-acting beta-2 agonists?

Case

A two-year-old boy presents to your office with a cough of three weeks duration. It is worse at night and with activity.

On further history, you discover the patient has prolonged cough following upper respiratory infections (URIs). He has been known to wheeze in the past.

He has never been diagnosed with asthma, but has been told he had bronchitis twice and put on antibiotics both times. He has never previously been treated with inhalers.

The child also has a history of eczema and his mother has asthma. She smokes and there are two cats in the house. His grandfather baby sits. There are cats and a wood-burning stove at the grandfather's home.
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by the presence of lymphocytes, eosinophils and mast cells. This airway inflammation has been associated with an increase in airway responsiveness to various stimuli and pathologic airway changes, including airway edema, airway smooth muscle hypertrophy and mucous gland hypertrophy. Long-term control has been associated with decreased airway reactivity, partial or complete regeneration of airway pathology and, of course, improved clinical outcome.

Table 1
The asthma history

<table>
<thead>
<tr>
<th>Symptoms</th>
<th>Cough, wheeze, shortness of breath, chest tightness</th>
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</thead>
<tbody>
<tr>
<td>Pattern of symptoms</td>
<td>Perennial, seasonal or both; continual, episodic or both; onset, frequency; diurnal variation, especially nocturnal and upon waking in early morning</td>
</tr>
<tr>
<td>Precipitation/ aggravating factors</td>
<td>Viral illness, environmental allergens (i.e., mold, dust mites, animal dander, pollen), exercise, environmental change, irritants (i.e., smoke, air pollutants), emotional (i.e., crying, laughing), drugs (i.e., acetylsalicylic acid, beta blockers), foods, change in weather</td>
</tr>
<tr>
<td>Development and treatment of disease</td>
<td>Age of onset, progression of disease, present/previous management, oral corticosteroids in past</td>
</tr>
<tr>
<td>Family history</td>
<td>Asthma, allergies, hayfever, eczema</td>
</tr>
<tr>
<td>Social history</td>
<td>Level of education, social support, employment, drug plan</td>
</tr>
<tr>
<td>Environmental history</td>
<td>Age of home, heating system, presence of wood-burning stove, carpet, mold/mildew, smoking, pets, environmental history of daycare/school/grandparents</td>
</tr>
<tr>
<td>Impact of asthma on patient</td>
<td>Emergency room, walk-in clinic visits; intensive care unit admissions; missed school days; history of nocturnal waking; impact on family routines; economic impact</td>
</tr>
<tr>
<td>Assessment of patient's/ parent's understanding of illness</td>
<td>Patient's/parent's knowledge of illness and use of medication, perception and beliefs regarding effects of long-term medication, level of family support</td>
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</tbody>
</table>
How do I diagnose asthma in kids?

The diagnosis of asthma in children is difficult. Unfortunately, spirometry in the young child is difficult, making the diagnosis a clinical one. Thus, a good history and physical examination are key in the diagnosis (Table 2). Often, the diagnosis may not be made at the first visit, but after recurrent symptoms are present. The Canadian Asthma Consensus Report states that establishing the diagnosis of asthma in the young patient depends on severe episodes of wheezing; wheezing after one year of age; more than three episodes of wheezing in a given year; a family history of asthma or atopy; a personal history of asthma or atopy; maternal smoking; clinical improvement after anti-inflammatory treatment; chronic cough (especially nocturnal or associated with exercise); and wheezing when viral etiology is unlikely. The likelihood of a diagnosis increases with the number of factors present.

Once a diagnosis of asthma is established, it is important to evaluate the severity of illness. This will help you in deciding a treatment regimen. It is best to break up asthma severity into categories of mild, moderate and severe. This requires careful assessment of symptom frequency and lung function. The history should include a comprehensive evaluation of symptom frequency during the day and night, and during exercise. The pattern of symptom frequency should be established. The symptom history should include cough, chest tightness, shortness of breath and wheezing. The pattern and frequency of beta 2-agonist use should also be established. The
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family history, past medical history and environmental history are all important in the diagnosis and when establishing a treatment plan.

The 1997 National Asthma Education and Prevention Program Expert Panel Report 2 helped define asthma severity as follows:3

Mild asthma: Children who have mild asthma experience daytime symptoms two times a week or less, and nighttime symptoms two times or less per month. Symptomatic episodes are brief and resolve spontaneously with the use of beta agonists. In between episodes, the patients are asymptomatic and have normal pulmonary functions (forced expiratory flow in one second [FEV1] and peak expiratory flow [PEF] greater than 80% of that predicted).

Moderate asthma: Children whose frequency exceeds those described in the mild category fit into the moderate category. They have night-time symptoms and their ability to perform daily activities may be limited. In between episodes they are symptomatic. Their PEF is 60% to 80% of that predicted. These patients are likely to have a greater degree of airway inflammation and hyperactivity. They will need to receive daily medication to control their asthma.

Severe asthma: The child with severe asthma uses beta agonists frequently, has a history of multiple hospital visits and uses prednisone. They have night-time symptoms and are limited in their ability to perform daily activities, which includes attending school. They may have had a prior near fatal episode. The asthma severity categories are summarized in Table 3.2

What asthma medications are available and what are their roles?

Before we begin on the management of asthma, let’s review the medications used in the current management of asthma.
**Beta 2-agonists:** Short-acting beta 2-agonists relax smooth muscles and promote increased airflow. Therefore, these medications are the first-line choice for treating acute asthma symptoms and exacerbations. Short-acting beta 2-agonists are also excellent for preventing exercise-induced symptoms. They are used on an as needed basis only. When the use of short-acting inhaled beta 2-agonists exceed three times a week (in addition to their once daily use for exercise-induced symptoms), however, regular anti-inflammatory medications should be added to the treatment regimen.

Long-acting beta 2-agonists are beneficial when added to inhaled glucocorticosteroids. They help address exercise-induced symptoms for longer periods than do shorter-acting beta 2-agonists.\(^4\) Patients receiving moderate doses of inhaled steroids with inadequate symptom control may benefit from adding a long-acting beta 2-agonist rather than increasing the dose of the inhaled steroid.\(^5,6\) Long-acting beta 2-agonists should be used as an add-on therapy, and are not recommended for relief of acute symptoms or use in absence of inhaled anti-inflammatory medications.

**Inhaled glucocorticosteroids:** These represent the most effective long-term therapy available. Inhaled glucocorticosteroids are well tolerated and safe at recommended doses.\(^7\) Local adverse effects include candidiasis. Patients should rinse their mouth out after inhaling steroids to reduce oropharyngeal deposition and possible oral candidiasis. Patients using low-to-moderate doses of inhaled corticosteroids for short periods of time are not at risk for adrenal insufficiency. The risk of cataracts appears to be low and routine screening is not recommended.

There is a slight decrease in growth velocity with regular use of inhaled glucocorticosteroids, but patients usually grow to reach average adult height.\(^8,9\) On the other hand, childhood asthma itself appears to be associated with delayed maturation and longer periods of reduced growth prior to puberty. These delays, however, do not appear to compromise the attainment of final adult height.\(^10,11\) Periodic growth monitoring is recommended in children using inhaled glucocorticosteroids.
Inhaled glucocorticosteroids can be divided into low, moderate and high doses. The initial dose should be 200 mcg to 1,000 mcg of beclomethasone equivalence. Once results are achieved, the dose should be reduced to the minimum dose required to maintain control. At the first sign of exacerbation, the inhaled steroid should either be added or increased two- to four-fold. This increase should be continued for the duration of the exacerbation but, once control is achieved, the dose should be reduced to the minimum required level to maintain control. For inhaled steroids, a twice-daily dose is effective, even at high doses. This also ensures compliance. Oral prednisone may be added if the exacerbation is severe.

**Leukotriene receptor antagonists (LTRAs):** Leukotrienes are potent mediators released from mast cells, eosinophils and basophils. They contract airway smooth muscle, increase secretions, and attract and activate inflammatory cells. The LTRA montelukast has been shown in clinical studies to be effective and generally well tolerated in six- to 14-year-old children with asthma. It was also found to be well tolerated in children between ages two to five years. The same study found clinical improvement in parameters of asthma control, as well as lower day-time and night-time scores, reduced need for oral corticosteroid rescue and an increase in days without asthma. Montelukast is available in a chewable tablet form for children less than 14 years of age.

Current recommendations suggest using LTRAs as an alternative to increased doses of inhaled glucocorticosteroids beyond moderate doses. They also have been shown to reduce exercise-induced symptoms. The use of LTRAs as first-line anti-inflammatories in place of inhaled glucocorticosteroids is being studied, but there is insufficient evidence at this time to recommend first-line use. For patients who cannot or will not use inhaled glucocorticosteroids, however, LTRAs should be the primary treatment of choice.

**Case discussion**

You diagnose this child with asthma. You educate the family about environmental clean-up at home and at the grandfather’s home. The family should also be educated about asthma and offered a referral to an asthma nurse educator. You start the child on an inhaled, short-acting beta 2-agonist metered dose inhaler (MDI) with spacer on a demand basis. You teach the family how to properly use the spacer. The child should be started on an inhaled glucocorticosteroid at moderate doses via MDI and spacer (fluticasone 125 µg, two puffs twice daily for a total of 500 µg/day). You review the side effects and safety of the medication to ensure compliance. The family is remind-
ed to rinse out the child’s mouth after the use of the inhaled steroid to prevent oral candidiasis. Finally, you arrange followup in four weeks.

Four weeks later, the family states the child is 100% better. He no longer has his cough, sleeps through the night without any symptoms and runs around without coughing, easily keeping up with the other children. He rarely uses his beta 2-agonist. The family has done a fair amount of environment clean-up, but the child’s mother still smokes in the car. You are very pleased.

You now advise the family to wean his inhaled steroids to one puff, twice daily (250 µg/day of fluticasone). The family does this and the child continues to do well, but when they tried to wean further his symptoms returned. You decide to keep him at one puff, twice daily of fluticasone (250 µg/day). This, you remember, is a low-to-moderate dose of an inhaled glucocorticosteroid. You again educate the family about the safety of the medication and about proper use of the spacer device, and caution the mother again about smoking in the car. The child is now well controlled on low-to-moderate doses of inhaled glucocorticosteroids. The family knows to use the short-acting beta 2-agonists on demand. You give them a written asthma action plan.

If the child begins to have symptoms of asthma, or at the first sign of a viral illness, you advise the family to use his short-acting beta 2-agonist on demand every four hours. You advise the family to increase the dose of the fluticasone two- to four-fold for at least two weeks, until the symptoms have resolved, and then to wean the dose back to the maintenance dose. The family is comfortable with the plan. You book regular follow-up meetings and advise them to return if the asthma gets out of control. You note that if, in the future, the child needs higher doses of inhaled glucocorticosteroids to maintain control, you would consider other therapy with either long-acting beta 2-agonists or LTRAs. Finally, you also note that when the child is able, you will prescribe him a peak flow meter for him to use at home.
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References

One depression can lead to another.