Summary

Asthma is a common disease, affecting 3-5% of the population, and 3 basic concepts are introduced here:
(1) Asthma is an inflammatory disease;
(2) there is a shift in treatment and (3) a management plan should be worked out with the patient and then written down. Several aspects are dealt with in a practical, helpful way, e.g. the correct diagnosis, allergy, asthma as an inflammatory disease, the aims of treatment, drug evaluation, a few common failings in management and how to address them.

Introduction

Asthma is a common disease affecting approximately three to five percent (3-5%) of the population. Despite this, the primary mechanisms have yet to be identified. As a result, there is no single criterion for defining asthma that can be applied in the clinical setting. Asthma is a chronic disease with acute exacerbations and severity that can resolve spontaneously, or with therapy, it is airway inflammation and airway hyperresponsiveness to a variety of stimuli.

Diagnosis

There is episodic coughing, dyspnoea, wheezing and chest tightness, alone or in combination. When the disease is active, patients will awaken at night or early in the morning with these symptoms. Nocturnal awakening is such a common feature of asthma that the absence from the history puts
doubt on the diagnosis.\(^3\) Wheeze is a hallmark of asthma, but it is not specific for asthma. All that wheezes is not asthma. Chronic obstructive airway disease wheezes as well as left heart failure and other rarer causes. Recurrent episodes of wheezing are almost always a hallmark of asthma.\(^3\)

To establish the presence of asthma, one's clinical impression must be confirmed objectively with a measurement of airflow.

**Therefore:** measure airflow rates: FEV1 and/or Peak flow

*If airflow is reduced:*
- Give \(\beta_2\)-Agonist (2 puffs)
- Repeat measurement after 15 minutes

*If more than 15% improvement in FEV1:* then the diagnosis of reversibility is confirmed.

**Problem:** You may get chronic airflow limitation in which the improvement may only occur after weeks or months of treatment and not immediately.

*If airflow is normal:* do provocation tests in a laboratory if asthma is suspected.\(^4\)

**Allergy in Asthma**

A critical issue concerning the pathogenesis of asthma is the role of allergens. It has been suggested that many patients with asthma have an atopic component. An important link between asthma and allergic factors, such as a history of atopy or elevated IgE levels, has long been recognised. Both genetic and environmental factors may contribute to IgE production.\(^3\)

**Reversibility**

In the vast majority of patients, the clinical course is characterised by exacerbations and remissions. In a small number of patients data suggest that irreversible changes may develop over time, as a function of the duration and severity of asthma.

**Asthma as an inflammatory disease**

Three features of asthma deserve special mention:

1. **Airway obstruction**
   - This is responsible for many of the clinical manifestations.
   - Airway obstruction is reversible in the majority (but not in everyone).

2. **Airway inflammation**
   - The intensity of the inflammation relates to the severity of the disease. Bronchial inflammation in varying degrees are present in the airways of asthmatics, including those with mild disease; it is even present in asymptomatic patients.\(^6\)

3. **Increased airway responsiveness (Hyperactivity)**
   - Airway inflammation and hyperresponsiveness are not unique to asthma. They are also found alone or together in other forms of airway disease, such as cystic fibrosis, chronic bronchitis and atopic rhinitis.

**Diagram 1. Pathophysiology ("Vicious Cycle")**

- Inflammation
- Bronchial obstruction
- Release of chemical mediators
- Bronchial hyperresponsiveness

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If the patient does not mention nocturnal awakenings, the diagnosis is probably not asthma.
Increased bronchial hyperresponsiveness is related to the degree of inflammation in the airways. Bronchial Hyperresponsiveness is non-specific and can be induced by: * Cold air, ozone, smoke SO2, laugh, sneeze and various other stimuli. (See Diagram 2)

**Aims of treatment**

There are five aims of treatment which attempt to address five common failings in management (they are given in brackets.)

1. Make the diagnosis [Under diagnosis]
   Like hypertension, asthma is often a “silent” disease, which often results in missed or delayed diagnosis.
2. Wipe out symptoms [Under treatment]
3. Restore normal lung function [Under measurement]
4. Reduce risk of an acute attack [Under prevention]
5. Provide the patient with an “action plan” [Under communication].

There is a world-wide trend towards the provision of guidelines for treatment and self management by the patient. One has to acknowledge that many aspects of diagnosis and treatment remain at best, educated guesses and that therapeutic regimens must be highly individualised.3

**Choice of therapy**

1. An anti-inflammatory drug: This should be the first-line of therapy. The primary aim is to reduce the underlying disease: chronic inflammation.3
2. ß-adrenergic agonists and theophylline: Should be used for symptomatic relief only.
3. Oral steroids: This may be necessary to control disease if first line fails.
4. Guidelines: Every doctor should use these, as many of them are available. South Africa has its own guidelines, and these should be consulted.7
5. Environmental control: This is critical. Measures to reduce exposure to allergens and irritants must be used.
6. Patient education: An educated patient is better able to achieve and maintain asthma control. This should be an ongoing process.

Here follows an example of a management plan:3

**Management plan [“Action”]**

The patient measures his own peak flow in the morning and at night.

* Target peak flow:
  1. Establish the patient’s maximum peak flow.
  2. 90 % of normal is calculated and this value is used as the target peak flow.
* If peak flow is:
  1. less than 90% of normal, or

**Diagram 2. The Phases of Asthma**

The early phase is one of bronchospasm. The late phase is one of inflammation.

Provide the patient with a written action plan.

Patient education is an ongoing process and is very important.

All that wheezes is not asthma.
Aspects of Asthma

(ii) varies with more than 25% between the morning and evening measurement, then: increase inhalation steroids (double the dose) for 1 to 2 weeks, and if peak flow returns to normal, reduce the dose. If no improvement, or if peak flow less than 60% of normal:

Start Oral steroids: The dose and duration of the course of steroids should be discussed with the patient.

Anti-inflammatory agents

The focus of treatment is now on prophylaxis and treatment of the basic inflammatory component of asthma rather than on the symptomatic broncho-constrictive aspect. Symptom modification takes a back seat to disease modification. The clinical benefits of regular inhaled corticosteroids have been well described. All patients with asthma (maybe other than mild, episodic) should receive inhaled anti-inflammatory medication.

Oral steroids

Short courses of oral steroids may be needed, regardless of diagnosed severity of asthma or the use of other asthma medication.

Dose: 0.5 - 1 mg/kg/day

Principles:
(1) Do not be afraid of short courses (2 weeks)
(2) Do not be afraid of a high dose (1 mg/kg/day)
(3) Do not be afraid to stop rapidly if used for less than two weeks

β-Stimulants

Still the mainstay of symptomatic treatment.* Still effective for relief of symptoms.

Other bronchodilators

Theophylline:
(i) Theophylline is a less effective bronchodilator than the β-agonists.
(ii) There is a trend now to start theophylline later in the treatment plan to provide additional control of symptoms if aerosol therapy [β-agonists and corticosteroids] is insufficient.

Anticholinergic Agents:
Ipratropium bromide provides varying degrees of bronchodilation.

Conclusion

Three concepts are thus emphasised in this article

(1) Asthma is an inflammatory disease of the airways.
(2) There is a shift in treatment. Symptomatic relief is necessary, but a move from symptom relief to inflammation prevention and diminishing of the inflammation should be the target.
(3) A management plan should be explained to the patient and written down.

Reducing the severity of the inflammatory process appears to be a reasonable goal in order to reduce long-term morbidity in asthma. It is hoped that these simple guidelines will help to achieve just that.

References