

More about... Asthma

Asthma versus COPD: What is the difference?

M L WONG, MB BCh, DCH (SA), FCP (SA),
FCCP, FRCP (Lond)

Principal Specialist and Head, Division of Pulmonology, Chris Hani Baragwanath Hospital, Johannesburg
Senior Lecturer, School of Clinical Medicine, Faculty of Health Sciences, University of the Witwatersrand, Johannesburg

Both asthma and chronic obstructive pulmonary disease (COPD) are chronic diseases characterised by airflow obstruction, and result from underlying airway inflammation. Whereas the airflow obstruction is reversible in asthma, in COPD it is at best only partially reversible. The inflammation in asthma is mediated by T-lymphocytes and eosinophils, but in COPD the neutrophil is the dominant inflammatory cell. Long-standing, poorly controlled asthma may evolve to fixed airflow obstruction, and thus be indistinguishable from COPD. Conversely, some patients with COPD may have partial (but significant) reversibility and respond to corticosteroids, and their condition may therefore resemble asthma to some degree.

There are no bedside or commercially available tests that directly and reliably measure the nature of airway inflammation in these two conditions. To differentiate asthma from COPD one is dependent on the history, clinical examination during the stable phase, and demonstration of the degree of airflow reversibility in response to administration of a short-acting β_2 -agonist and/or corticosteroid.

History

Asthma usually presents in childhood; however, adult-onset asthma is not uncommon. Frequently there is a family history of atopy. COPD presents in adulthood, usually in persons >40 years of age. In most cases, there is a strong history of smoking (cigarettes, cannabis).

The usual symptoms are cough, wheeze and dyspnoea. In asthma the cough is minimally productive and typically worse at night. Patients with COPD are more likely to have a productive cough, usually of mucoid sputum, which is worse in the morning.

In both conditions exacerbation of symptoms is often worsened by infections. Viral infections, in particular, are common precipitants, but about half of COPD exacerbations are related to bacterial infections. Other causes of acute asthma attacks are exposure to dust, smoke, pollen, animal dander, drugs (e.g. β -blockers, aspirin), exercise, cold weather, and occupational exposures. Acute exacerbations of COPD may be precipitated by air pollution, cold weather and pulmonary thrombo-emboli.

Clinical features

During acute exacerbations it may be difficult to distinguish asthma from COPD. In both diseases patients show signs of respiratory distress with hyperinflation of the chest. Audible wheezing or a silent chest may be present.

In the stable state, examination of the patient with asthma is unremarkable. Evidence of atopic disease (e.g. allergic rhinitis) may be present. However, the COPD patient frequently demonstrates pursed lip breathing, tachypnoea at rest, chest hyperinflation, decreased intensity of breath sounds, and crackles on auscultation. There may also be evidence of chronic hypoxaemia and pulmonary hypertension (plethora, cyanosis, cor pulmonale).

Lung function tests: Peak expiratory flow (PEF), spirometry

Asthma patients should achieve normal lung function, whereas COPD patients demonstrate persistent airflow obstruction ($FEV_1/FVC < 70\%$ on spirometry) even when at their best.

All patients with evidence of airflow obstruction (on PEF measurement or spirometry) suspected to be caused by asthma or COPD should undergo assessment for airflow reversibility. This should be done by the administration of a short-acting β_2 agonist, e.g. salbutamol 200 - 400 μg (via metered-dose inhaler or nebuliser), followed by repeat PEF measurement or spirometry 20 minutes later. Clinically significant reversibility is indicated by a PEF measurement that increases by 20% from baseline, or by spirometry where the FEV_1 increases by at least 12% from baseline together with an absolute increase of 200 ml or more. However, it must be noted that these criteria are not useful if the baseline

value is only mildly abnormal and may erroneously lead to underestimation of reversibility.

If this post-bronchodilator lung function does not normalise, patients should be subjected to a trial of corticosteroid therapy (prednisone 40 mg daily for 2 weeks, or inhaled budesonide 400 μg bd or equivalent for 6 weeks). Restoration of lung function to normal is in keeping with a diagnosis of asthma, but partial improvement is more indicative of COPD.

If the patient has normal lung function at consultation, but has symptoms of asthma, demonstration of exaggeration of the normal diurnal variation of PEF is useful. Patients should record PEF measurements in the morning and evening. Variability of >20% is highly suggestive of asthma. Another simple test to demonstrate airway hyperresponsiveness is to determine PEF measurements before and 10 minutes after 6 minutes of exercise. A decrease in PEF of 20% or more (or a 15% fall in FEV_1) precipitated by exercise is also suggestive of asthma. It is important to be aware that other causes of hyperresponsiveness do exist. These include recent viral lower respiratory tract infections in patients without asthma (for up to 6 weeks).

Clinical importance of distinguishing asthma from COPD

Untreated or inadequately treated airflow obstruction results in avoidable morbidity and impaired quality of life. Early detection of COPD affords an opportunity to encourage smoking cessation and reduce long-term consequences. All asthma patients must be prescribed inhaled corticosteroid therapy. However, only COPD patients who display a clinically significant response to corticosteroids or who have three or more acute exacerbations per year have been shown to benefit from maintenance inhaled corticosteroid therapy. Unnecessary prescription of these drugs results in increased morbidity and unwarranted health care costs.

Bibliography

COPD Guideline Working Group of the South African Thoracic Society. Guideline for the management of chronic obstructive pulmonary disease (COPD): 2004 revision. *S Afr Med J* 2004; 94(7): 559-575.

Lalloo U, Ainslie G, Wong M, et al. Guidelines for the management of chronic asthma in adolescents and adults. *SA Fam Pract* 2007; 49(5): 19-31.

Asthma in special situations

MOHAMED SABEER ABDOOL-GAFFAR, MB ChB, FCP (SA), FCCP
Pulmonologist, Kingsway Hospital Medical Centre, Amanzimtoti

Asthma during pregnancy

Asthma is one of the most common medical conditions and the most common respiratory disorder complicating pregnancy.¹ It has a variable course during pregnancy: one-third of patients improve, one-third remain unchanged and one-third experience deterioration of their symptoms.² Patients with more severe pre-existing asthma tend to have more frequent exacerbations during pregnancy.^{2,3} It is unclear why the course differs in different patients. Also, the course may vary in the same patient in different pregnancies.¹ Asthma tends to return to the pre-pregnancy level of severity within 3 months of delivery.⁴ Poorly controlled asthma is associated with poorer outcomes in pregnancy.^{1,3} There is an increased risk of perinatal mortality, low-birth-weight infants, preterm delivery and pregnancy-induced hypertension.^{1,3} Some of the mechanisms proposed for these effects are maternal hypoxia, inflammation, medication, smoking and altered placental function.¹

Improved control of asthma during pregnancy, including prevention of exacerbations, is associated with improved pregnancy outcomes.¹ The use of inhaled corticosteroids to control maternal inflammation has been associated with better outcomes.¹ In addition to medical treatment it is also important to avoid triggers. The pharmacological management of asthma is the same for the pregnant and non-pregnant patient.³ There are extensive data supporting the safety of major drug classes used to treat asthma in pregnancy.¹ The stepwise use of inhaled, short-acting β_2 agonists, inhaled corticosteroids and inhaled long-acting β_2 agonists as per the current SATS guideline is recommended.⁵ Acute exacerbations of asthma can also be handled according to standard management during the non-pregnant state.

Asthma in the elderly

Asthma affects approximately 4 - 8% of people over the age of 65 years⁶ and 7 - 9% of individuals over the age of 70.⁷ However, the diagnosis is frequently overlooked and undertreated.⁷ There is a low index of suspicion for asthma in older patients, and the symptoms are often attributed to other causes such as congestive cardiac

failure and chronic obstructive pulmonary disease (COPD).⁷ There are some differences between elderly and younger asthmatics: in the elderly symptoms are often persistent rather than intermittent, atopy is less common, concomitant medications are frequently used and may precipitate asthma, and side-effects of medications are common.

Asthma is more severe in the elderly.⁸ The diagnosis can be confirmed by a careful history and clinical examination, followed by office spirometry before and after inhaling a bronchodilator. The absence of an acute bronchodilator response should not rule out a diagnosis of asthma in the elderly.⁹ Methacholine challenge testing may be useful in this situation, and a normal diffusing capacity will rule out emphysema in current and ex-smokers.⁹ Management of the elderly asthmatic does not differ from that of a younger patient, except that some of these patients, e.g. those with both asthma and COPD, may be more responsive to anticholinergics and less responsive to inhaled corticosteroids. Side-effects of anti-asthma drugs are more common. One must also consider frequent co-morbidity and drugs used to treat these conditions, which may adversely affect asthma.³

Asthma and GORD

It is widely accepted that there is an association between gastro-oesophageal reflux disease (GORD) and asthma. However, the relationship between the two diseases remains controversial.¹⁰⁻¹² A recent meta-analysis concluded 'that there is a significant association between GORD and asthma, but a paucity of data on the direction of causality'.¹⁰ The prevalence of GORD in asthma patients varies from 30% to 90%, depending on the definition and methods used.^{10,11} The two postulated mechanisms for exacerbation of asthma are: damage to the airway by acid reflux (reflux theory), or by bronchoconstriction resulting from stimulation of vagal nerve endings in the oesophagus (reflex theory).¹⁰ A Cochrane Review Abstract concluded that treatment of GORD in asthmatic patients did not result in overall improvement of asthma symptoms.¹² However, the author did concede that there may be subgroups of patients who may benefit, but that it is difficult to identify responders.¹² In addition, 'despite the enormous volume of literature that exists on the subject, there is a shortage of high-quality data'.¹⁰ Therefore, a pragmatic approach is recommended in the current SATS guidelines.⁵ In any patient with difficult-to-treat asthma one should consider the presence of GORD, recognising that many patients with GORD may be asymptomatic. Both diseases should be treated appropriately.

References

- Murphy VE, Gibson PG, Smith R, Clifton VL. Asthma during pregnancy: mechanisms and treatment implications. *Eur Respir J* 2005; 25: 731-750.
- Gluck JC. The change of asthma course during pregnancy. *Clin Rev Allergy Immunol* 2004; 26(3): 171-180.
- Schatz M. Special considerations in allergy and asthma. Pregnancy and geriatrics. *Clinical and Applied Immunology Review* 2001; 1: 225-228.
- Gluck JC, Gluck PA. The effect of pregnancy on the course of asthma. *Immunology and Allergy Clinics of North America* 2006; 26(1): 63-80.
- Lalloo U, Ainslie G, Wong M, et al. Guidelines for the management of chronic asthma in adolescents and adults. *South African Respiratory Journal* 2007; 13(4): 145-160.
- Kitch BT, Levy BD, Fanta CH. Late onset asthma: epidemiology, diagnosis and treatment. *Drugs Aging* 2000; 17(5): 385-397.
- Braman SS. Asthma in the elderly. *Clin Geriatr Med* 2003; 19: 57-75.
- Quadrelli S, Roncoxon A. Features of asthma in the elderly. *J Asthma* 2001; 38(5): 377-389.
- Enright PL. The diagnosis and management of asthma is much tougher in older patients. *Current Opinion in Allergy and Clinical Immunology* 2002; 2(3):175-181.
- Havemann BD, Henderson CA, El-Serag HB. The association between gastro-oesophageal reflux disease and asthma: a systematic review. *Gut* 2007; 56: 1654-1664.
- Vincent D, Cohen-Jonathan AM, Lepout J, et al. Gastro-oesophageal reflux prevalence and relationship with bronchial reactivity in asthma. *Eur Respir J* 1997; 10: 2255-2259.
- Gibson PG. Gastro-oesophageal reflux treatment for asthma in adults and children. *Cochrane Review Abstracts*. Posted 07/01/2007.

Exercise-induced asthma

SHARON KLING, MB ChB, DCH (SA), FCPaed (SA), MMed (Paed)

Senior Specialist, Department of Paediatrics and Child Health, Stellenbosch University and Tygerberg Children's Hospital, Tygerberg

Definition

Exercise-induced asthma (EIA) is defined as 'the condition in which exercise induces symptoms of asthma in patients who have asthma'.¹ The question is whether EIA is a distinct phenotype of asthma or whether it is just another trigger of symptoms in asthmatic patients. The authors of the AAAAI Report¹ do not view EIA and asthma as separate conditions. They use the term 'exercise-induced bronchospasm' (EIB) to describe airway obstruction after exercise without consideration of underlying chronic asthma.

More about...

Pathogenesis

EIA is caused by changes in airway physiology due to hyperventilation after exercise.¹ Two hypotheses are postulated: the hyperosmolarity theory and the airway-rewarming theory.^{1,2} The first theory holds that evaporation of water from the airway during exercise causes an increase in osmolarity with subsequent inflammatory mediator release, bronchoconstriction and airway oedema. The airway-rewarming theory proposes that initial airway cooling and subsequent rapid rewarming cause reactive vascular engorgement and airway wall oedema.

Prevalence

The prevalence of EIA varies considerably. Between 50% and 90% of people with persistent asthma will experience EIB after an appropriate exercise challenge. It affects 5 - 20% of the general population and 10 - 50% of elite athletes, being highest in winter sports competitors. The wide variation in these figures is attributable to the different populations surveyed and the method used to identify EIA.

Clinical presentation

The clinical features of EIA are cough, wheeze and tight chest after strenuous exercise of about 6 - 8 minutes. In elite athletes cough appears to be the most common symptom. Symptoms may not present until after the exercise if the latter lasts for a longer time.³ Athletes may 'run through' their asthma: the symptoms decrease if exercise intensity is reduced, but frequently recur after exercise is discontinued. Subtle presentations of EIA include abdominal pain, muscle cramps, fatigue, dizziness, or 'being out of shape'.³

Diagnosis

This depends on the type of patient presenting with symptoms. The average school child or adult recreational athlete tends to present with a classic cough, a wheeze and shortness of breath after exercise, but children may not recognise the symptoms as being those of asthma and may surmise that they are unfit.³ A careful history is required with regard to

the type of exercise and any associated breathing difficulty.

A detailed physical examination is necessary to diagnose concomitant allergic conditions and to rule out other causes of shortness of breath.

Lung function testing (spirometry) is an important diagnostic tool and should be performed before and after administration of a short-acting β_2 agonist. It is not necessary to perform exercise-challenge testing in the majority of school-age children and adult recreational athletes, provided their baseline FEV₁ is normal and they respond well to treatment.³ An FEV₁ <90% is suggestive of chronic, persistent asthma with an exercise-related exacerbation.

In elite, competitive athletes exercise-induced respiratory symptoms are usually poor predictors of EIA.¹ Treatment must not be given for a condition that does not exist. In these individuals an exercise challenge or a surrogate challenge should be done to evaluate bronchial hyperresponsiveness. The exercise challenge may have to be performed in the environment that usually causes the EIA-type symptoms.

Differential diagnosis

The differential diagnosis of EIA includes cardiac and respiratory conditions that can cause exertional dyspnoea, exercise-induced laryngeal dysfunction, gastro-oesophageal reflux, exercise-induced hyperventilation, and exercise-induced anaphylaxis. The following group of conditions may cause diagnostic difficulties in elite athletes: exercise-induced laryngeal dysfunction, which includes exercise-induced (paradoxical) vocal cord dysfunction, laryngeal prolapse, and laryngomalacia. These conditions most commonly present in young adult women with inspiratory stridor and throat tightness during maximal exercise, and resolve within 5 minutes of discontinuing exercise. This contrasts with EIA where the dyspnoea is expiratory, presents after exercise, and peaks 5 - 20 minutes after discontinuing exercise.¹

Treatment

Treatment of EIA involves the following:

- education – types of exercise (e.g. swimming); warming up before exercise to utilise the refractory period; avoidance of exercise in cold air or on high-pollen days
- pharmacotherapy
- follow-up.

The most effective drug for EIA is inhaled salbutamol (or another rapid-acting β_2 agonist) 2 - 4 puffs 15 minutes before exercise. If it does not prevent EIA, other diagnoses must be considered which may require regular anti-inflammatory therapy (inhaled corticosteroids) for asthma. Long-acting β_2 agonists will also block EIA, but tachyphylaxis and safety concerns mitigate against their use as monotherapy. Other options for EIA therapy are inhaled cromolyn or nedocromil, or leukotriene-receptor antagonists. Nasal symptoms impact on the severity of asthma associated with exercise; therefore allergic rhinitis must be adequately treated.

The performance of athletes may be enhanced by asthma drugs, which are prohibited both during and outside of competition. The medical practitioner has to certify the diagnosis of asthma, and the competitive athlete has to apply for therapeutic use exemption of asthma therapy from the South African Institute for Drug-Free Sports.⁴

References

1. Weiler JM, Bonini S, Coifman R, *et al.* American Academy of Allergy, Asthma & Immunology Work Group Report: Exercise-induced asthma. *J Allergy Clin Immunol* 2007; 119: 1349-1358.
2. Milgrom H. Exercise-induced asthma in the competitive athlete. In: Szefer SJ, Pedersen S, eds. *Childhood Asthma. Lung Biology in Health and Disease*. Vol. 209. New York: Taylor & Francis, 2006.
3. Storms WW. Asthma associated with exercise. *Immunol Allergy Clin N Am* 2005; 25: 31-43.
4. The South African Institute for Drug-Free Sports. *The Athlete's Guide to Drug-Free Sports*. 7th ed. Section 1: 8-10, 2008. [http://www.drugfreesport.org.za/pdf/The Athletes Guide to Drug-Free sport 2008-7 Edition.pdf](http://www.drugfreesport.org.za/pdf/The_Athletes_Guide_to_Drug-Free_sport_2008-7_Edition.pdf) (accessed 25 March 2008).