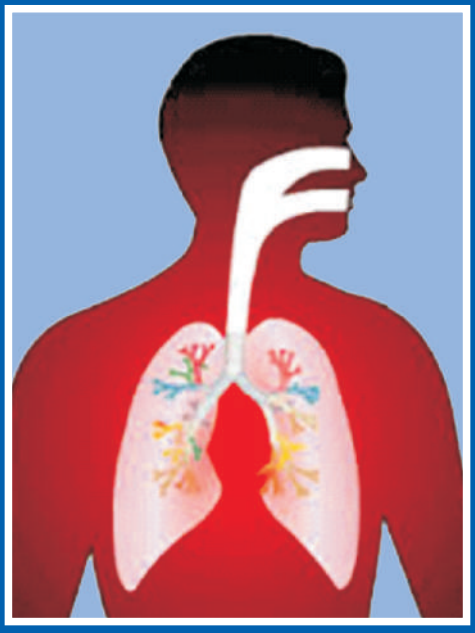
Republic of Zambia

Ministry of Health

NON-COMMUNICABLE DISEASES GUIDELINES



CHRONIC OBSTRUCTIVE AIRWAY DISEASES & ASTHMA

08

Republic of Zambia

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NON-COMMUNICABLE DISEASES GUIDELINES

CHRONIC OBSTRUCTIVE AIRWAY DISEASES & ASTHMA

Volume 8

CHAPTER EIGHT

A. ASTHMA:

Definition

Asthma is a chronic disease characterized by recurrent attacks of breathlessness and wheezing, which vary in severity and frequency from person to person.

Symptoms Include;

- wheezing,
- Coughing,
- chest tightness and,
- Shortness of breath.

Predisposing factors of Asthma

Probably due to a combination of environmental and genetic (inherited) factors.

Environmental factors include:

- Airborne allergens, such as pollen, animal dander, mold, cockroaches and dust mites
- Respiratory infections, such as the common cold
- Physical activity (exercise-induced asthma)
- Cold air
- Air pollutants and irritants, such as smoke
- Certain medications;
- including beta blockers, aspirin and other non-steroidal anti-inflammatory drugs
- Strong emotions and stress
- Sulfites, preservatives added to some types of foods and beverages
- Gastroesophageal reflux disease (GERD).
- Allergic reactions to some foods, such as peanuts or shellfish

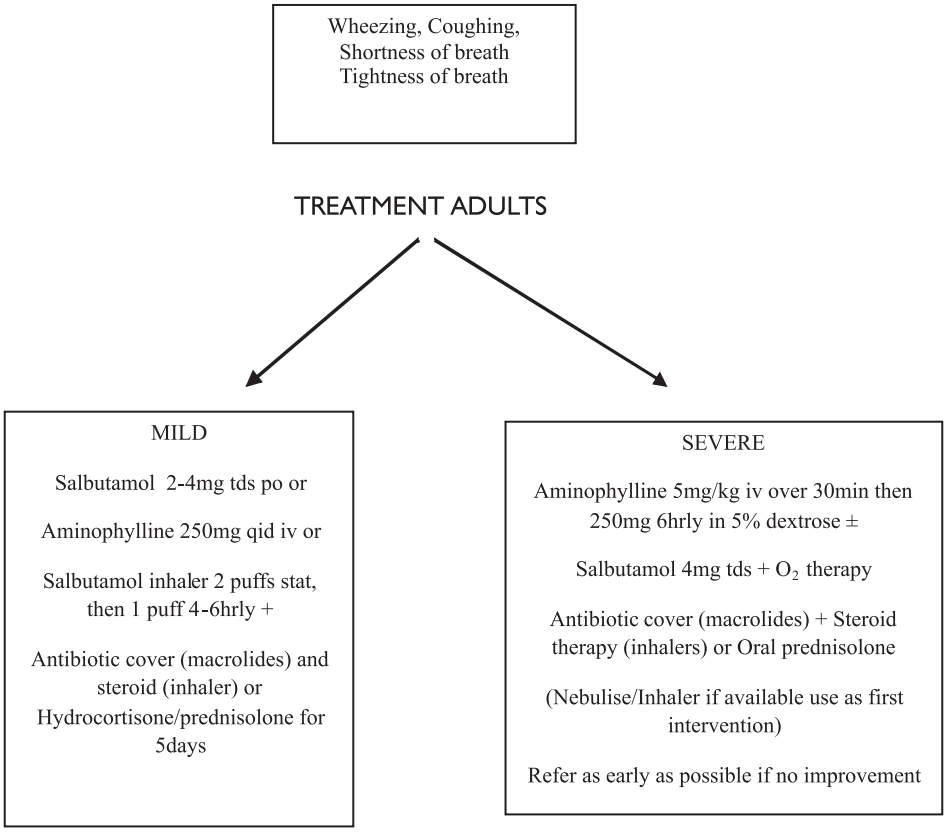
Genetic factors.

- Family History of Asthma: If a person has a parent with asthma, he or she is three to six times more likely to develop asthma than someone who does not have a parent with asthma.
- Airway Hyper reactivity: Not all people with airway hyper reactivity develop asthma, but in those who do have it, the airway hyper reactivity appears to increase the risk of asthma.
- Atopy: Atopy means allergic hypersensitivity that affects different parts of the body

that do not come in contact with allergens, substances that trigger the body's allergic reaction. Atopy can include eczema (atopic dermatitis), allergic rhinitis, allergic conjunctivitis, and asthma.

- Allergies Linked to Asthm: Allergies and asthma often coexist. Indoor allergies are a predictor of who might be at risk for an asthma diagnosis. Sources of other indoor allergens include animal proteins (particularly cat and dog allergens), dust mites, cockroaches, and fungi.

FLOW CHART FOR DIAGNOSIS AND MANAGEMENT OF ASTHMA AT HEALTH POST/CENTRE LEVEL



Patients who do not show response to above measures are to be referred within 24hrs

ASTHMA IN CHILDREN

Asthma can often be diagnosed on the basis of a patient's symptoms and medical history. Presence of any of these signs and symptoms should increase the suspicion of asthma

- Wheezing high pitched whistling sounds when breathing in and out especially in children.

Note: A normal chest examination does not exclude asthma.

History of any of the following:

- Cough worse particularly at night
- Recurrent wheeze
- Recurrent difficult breathing
- Recurrent chest tightness
- Symptoms occur or worsen at night, awakening the patient
- Symptoms occur or worsen in a seasonal patten
- The patient also has eczema, hay fever, or a family history of asthma or atopic disease

Risk factors (Symptoms occur or worsen in the presence of):

- Animals with fur
- Aerosal chemicals
- Changes in temperature
- Domestic dust mites
- Drugs (aspirin, beta blockers)
- Exercise
- Pollen
- Respiratory (viral) infections
- Smoke
- Strong emotional expression
- Symptoms respond to anti-asthmatic therapy
- Patients colds "go to the chest" or take more than 10 days to clear up.

Measurements of lung function provide an assessment of the

- severity,
- reversibility,
- variability of airflow limitation,
- help to confirm the diagnosis.

Assessment of severity

The severity of the exacerbation determines the treatment.

Severity of Asthma Exacerbations*				
	Mild	**Moderate**	**Severe**	**Life threatening**
Symptoms				
Breathless	Walking	Talking, Infant-softer shorter cry; Difficulty	At rest Infant stops feeding	
	Can lie down	feeding	Hunched forward	
		Prefers sittting		
Talks in	Sentences	Phrases	Words	
Signs				
Alertness	May be agitated	Usually agitated	Usually agitated	Drowsy or confused
Respiratory rate	Increased	Increased	Often >30min	
	Normal rates of breathing in awake children: Age Normal rate <2months <60/min 2-12months <50/min 1-5years <40/min 6-8years <30/min			
Accessory muscles & supraternal retractions	Usually not	Usually	Usually	Paradoxical thoraco-abdominal movement
Wheeze	Moderate, often only end expiratory	Loud	Usually loud	Absence of wheeze
Pulse	Increased	Increased	Increased	Bradycardia
	Guide to limits of normal pulse rate in children: Infants 2-12 months <160/min Preschool 1-2 years <120/min School age 2-6 years <110/min			
Pulsus paradoxus	Absent <10mmHg	May be present 10-25mmHg	Often present 20-40mmHg	Absence suggests respiratory muscle fatigue
Functional assessment				
PEF after initial bronchodilator % predicted or % personal best	Over 80%	Approx. 60-80%	<60% predicted or personal best	
SaO_2 %	>95%	91-95%	<90%	
*The presence of several parameters, but not necessarity all, indicates the general classification of the exacerbation.				

1st level and 2nd level

Initial treatment of acute asthma in children > 2 years

- Inhaled salbutamol via metered dose inhaler (MDI) and spacer is preferred for mild to moderate exacerbations.
- Salbutamol 100 mcg inhaler, 2-10 puffs every 10 to 20 minutes
- Salbutamol via nebuliser if $SaO_2 < 92\%$, 2.5 5 mg every 20-30 minutes
- Start with three back to back and then re-assess. If symptoms improve, taper down to hourly, two hourly, three hourly, four hourly MDI.
- If symptoms are refractory to β_2 agonist treatment Ipratropium bromide 250-500 mcg/dose mixed with salbutamol can be given

Steroid therapy

Oral steroids
- Give prednisolone early in the treatment of acute asthma attacks
- 20 mg in children 2-5 years
- 40 mg in children > 5 years
  (given for 3-5days)

Intravenous steroids
- For severe exacerbations or children who are vomiting.
- Hydrocortisone 4 mg/kg 4 hourly

Inhaled steroids
- No evidence of additional benefit
- Can be maintained in children already on long term therapy

Treatment of acute asthma in children less than 2 years
- β_2 agonist bronchodilator
- MDI via spacer is optimal drug delivery device for mild to moderate asthma

Steroid therapy
- Prednisolone 10 mg once daily for 3-5days

Ipratropium bromide
- Leads to some improvement in clinical symptoms
- Reduces the need for more intensive treatment
- If no improvement refer the patient to the hospital which has a HDC or ICU

3rd level

Second line treatment of acute asthma in children > 2 years

Continuing severe asthma despite

- Nebulised β_2 agonist
- Nebulised ipratropium bromide
- Oral steroids
- Showing signs of life-threatening asthma

Give
- IV salbutamol - 15 mcg/kg over 10 minutes then 1-5 mcg/kg/min infusion
- IV aminophylline - 5 mg/kg over 20 minutes then 1 mg/kg/hour infusion
- IV magnesium sulphate 40 mg/kg/day
- Child may need respiratory support in life threatening asthmatic attack.

When to refer
- Patients with the following clinical signs after 2 - 4 hours of repeated dosing with rapid-acting inhaled β 2-agonists:
- Worsening tachypnoea and subcoastal retractions
  Cyanosis
- Worsening O_2 saturations (where available)

FLOW CHART ON MANAGEMENT OF CHILDHOOD ASTHMA

SEVERE

- IV salbutamol - 15 mcg/kg over 10 minutes then 1-5 mcg/kg/min infusion + hydrocotisone 4 mg 4 houry
  or
- IV aminophylline - 5 mg/kg over 20 minutes then 1 mg/kg/hour infusion + hydrocotisone 4 mg 4 houry
  If no response (At tertiary level)
- IV magnesium sulphate 40 mg/kg/day + hydrocotisone 4 mg 4 houry

Symptoms

Wheezing,

Coughing,

MILD

- Inhaled salbutamol 100 mcg 2-10 puffs via metered dose inhaler (MDI) using spacer every 10 - 20 minutes + Predinesone 40 mg (>5 yrs), 20 mg (2-5 yrs), 10 mg (<2 yrs)

or

- nebilized salbutamol 2.5 mg (<5 yrs), 5 mg (> 5yrs) every 10 - 20 minutes + Predinesone 40 mg (>5 yrs), 20 mg (<5 yrs)

MODERATE

- Inhaled salbutamol 100 mcg 2-10 puffs via metered dose inhaler (MDI) using spacer every 10 - 20 minutes + Predinesone 40 mg (>5 yrs), 20 mg (2-5 yrs), 10 mg (<2 yrs)

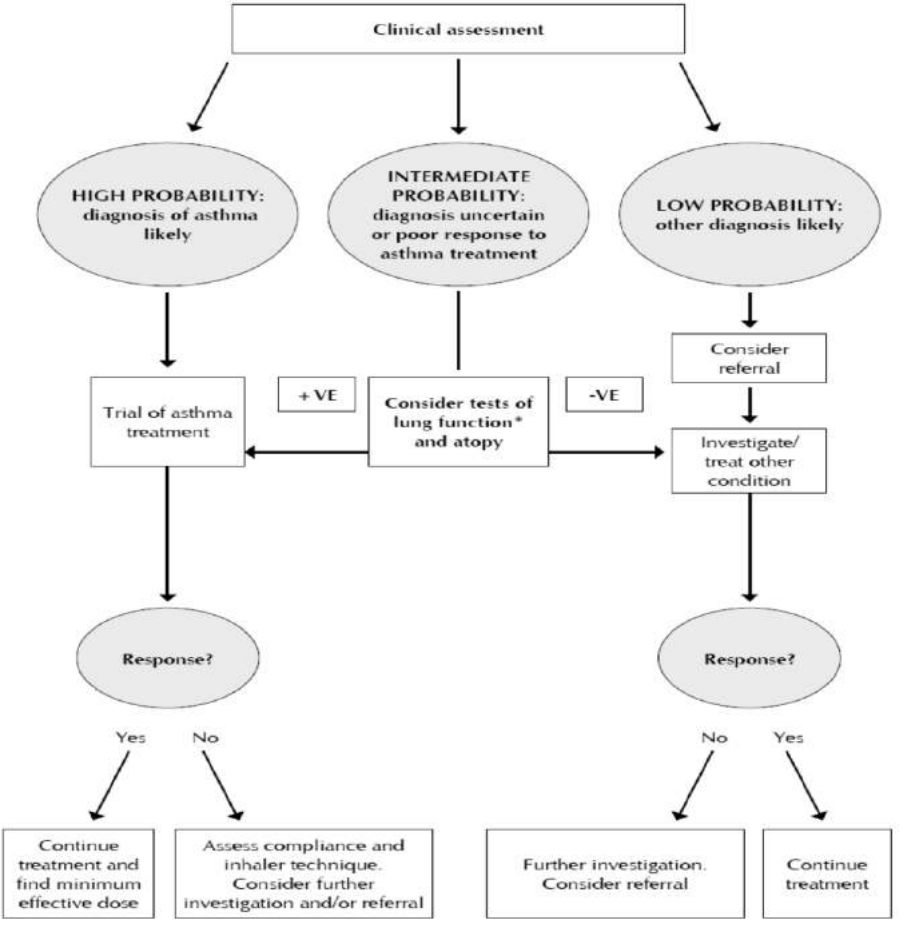
or

- nebilized salbutamol 2.5 mg (<5 yrs), 5 mg (> 5yrs) 10 - 20 minutes + Predinesone 40 mg (>5 yrs), 20 mg (<5 yrs)
- hydrocotisone 4 mg 4 houry for those who can not take oral steroids
- If no response give Ipratropium bromide nebilization

Referral criteria to a centre with high care or intensive care unit

- Patients with the following clinical signs after 2 - 4 hours of repeated dosing with rapid-acting inhaled β 2-agonists:
- Worsening tachypnoea and subcoastal retractions
- Cyanosis
- Worsening O_2 saturations (where available)

FLOWCHART ON PRESENTATION OF SUSPECTED ASTHMA IN CHILDREN



* *Lung function tests include spirometry before and after bronchodilator (test of airway reversibility) and possible exercise or methacholine challenge (tests of airway responsiveness). Most children over the age of 5 years can perform lung function tests.*

B. CHRONIC OBSTRUCTIVE PULMONARY DISEASE (COPD)

Definition

Chronic obstructive pulmonary disease (COPD) is a lung ailment that is characterized by a persistent blockage of airflow from the lungs. It is an under-diagnosed, life-threatening lung disease that interferes with normal breathing and is not fully reversible.

Stages of COPD

Chronic Obstructive pulmonary disease is divided into four stages;

STAGE	DESCRIPTION	MANIFESTATION	SYMPTOMS
I	Mild COPD	Mild airflow limitation	Chronic cough and sputum production
II	Moderate COPD	Worsening airflow limitation with shortness of breath	Worsened cough and shortness of breath developing on exertion
III	Severe COPD	Further air flow limitation	Greater shortness of breath, reduced exercise capacity, fatigue and repeated exacerbations.
IV	Very Severe COPD	Severe airflow limitation	Worsened shortness of breath with activity intolerance.

Epidemiology

Globally, non-communicable diseases (NCDs) are increasingly recognized as a major cause of morbidity and mortality. The World Health Report 2001 had indicated that NCDs account for almost 60% of deaths and 46% of the global burden of disease.Seventy-five per cent of the total deaths due to NCDs occur in developing countries.

The Zambian perspective: Non-communicable diseases are assuming alarming proportions in Zambia. Asthma and Chronic Obstructive Pulmonary Disease are also of major concern in the country. Despite there being no statistics about COPD and Asthma at national level, Ministry of Health is trying hard to strengthen data capture system at all levels of health care delivery with focus on non communicable diseases. These conditions affect different age groups in the population.

CAUSES OF COPD

Smoking

The primary cause of chronic obstructive pulmonary disease (COPD) is tobacco smoke (including second-hand or passive exposure). WHO estimates that in 2005 5.4 million people died due to tobacco use. Tobacco-related deaths are projected to increase to 8.3 million deaths per year by 2030.

RISK FACTORS OF COPD

Tobacco smoke

Worldwide, cigarette smoking is the most commonly encountered risk factor for COPD. Cigarette smokers have a higher prevalence of respiratory symptoms and lung function abnormalities, and a greater COPD mortality rate than nonsmokers. The risk for COPD in smokers is dose-related. Age at starting to smoke as well as passive exposure to cigarette smoke also predisposes someone to COPD. Smoking cessation has been associated with a reduction in the incidence of COPD.

Indoor air pollution

Wood, animal dung, crop residues and coal typically burnt in open fires or poorly functioning stoves lead to very high levels of indoor air pollution. Almost 3 billion people worldwide use biomass and coal as a source of energy for cooking, heating and other house hold needs.

Gender

Most of the earlier studies showed that COPD prevalence and mortality were greater among men than women. Studies from developed countries show that the prevalence of the disease is now almost equal in men and women which probably reflect changing patterns of tobacco smoking.

Genetic Factors

COPD is a polygenic disease and a classic example of gene-environment interaction. The genetic risk factor that is best documented is a severe hereditary deficiency of alpha-1 antitrypsin-4.

Inhalational Exposures

An individual may be exposed to a variety of different types of inhaled particles over their lifetime, which may contribute to the risk of COPD; but only tobacco smoke and occupational dusts and chemicals (vapours, irritants and fumes) are known to cause COPD on their own. Tobacco smoke and occupational exposures also appear to act additively to increase the risk of developing COPD.

Occupational dust and Chemicals

Occupational exposures are an underappreciated risk factor for COPD. These exposures include organic and inorganic dusts and chemical agents and fumes.

Outdoor air pollution
Air pollution from fossil fuel combustion, primarily from motor vehicle emissions in towns and cities is associated with decrement of the respiratory function.

Infections
Repeated infections; viral and bacterial may contribute to the pathogenesis of COPD, and may also play a significant role in acute exacerbations.

Nutrition
Malnutrition and weight loss can reduce respiratory muscle strength and endurance, apparently by reducing both respiratory muscle mass and the strength of the remaining muscle fibres.

MANAGEMENT OF CHRONIC OBSTRUCTIVE PULMONARY DISEASE

Chronic obstructive pulmonary disease (COPD) is a syndrome of progressive airflow limitation caused by chronic inflammation of the airways and lung parenchyma.[1] The primary physiological abnormality in COPD is an accelerated decline in the forced expiratory volume in one second (FEV_1) from the normal rate in adults over 30 years of age of approximately 30 ml per year to nearly 60ml per year.[2]

Essentials of diagnosis
- Primarily consisting of emphysema and chronic bronchitis
- Dyspnea or chronic productive cough or both are characteristic;
- COPD is nearly always a disease of heavy smokers (80 - 90%)
- Tachypnea, barrel chest, distant breath sounds, wheezes or rhonchi, cyanosis; clubbing unusual
- Hypoxemia and hypercapnia more pronounced with chronic bronchitis than with emphysema, whereas pulmonary hypertension is more common in patients with emphysema Hyperexpansion with decreased markings by chest radiography
- Airflow obstruction by spirometry (FEV1/FVC ratio < 0.70); reduced diffusing capacity (DLCO) in emphysema

TREATMENT:
- Stopping smoking is most important intervention
- Inhaled anticholinergic agents improve symptoms and decrease exacerbations (inhaled tiotropium may be superior to ipratropium)
- Long-acting β-agonists decrease exacerbations
- Chronic inhaled glucocorticoids may increase pneumonia risk; use only if patient has clear symptomatic improvement
- Supplemental oxygen for hypoxemic patients (Pao2 < 55 mm Hg or O2 saturation < 88%) reduces mortality
- For exacerbations, treat with bronchodilators, antibiotics, systemic glucocorticoids with taper over 2 weeks

PREVENTION AND CONTROL OF COPD

1. Primary Prevention

Population education on causes and risk factors, and symptomatology, with particular focus on tobacco smoking and indoor/outdoor air-pollution, using all possible media. Comprehensive public health actions focused on reducing tobacco use including health education, legislative and legal measures, fiscal measures and social action. These would also include measures to protect against passive smoking. Legislative, legal as well as social measures to reduce outdoor air pollution especially from automobiles and industries.

2. Secondary Prevention

Training and retraining of health care workers especially at the primary care level for keeping a high diagnostic suspicion and diagnosing the disease at the earliest. Provision of diagnostic (especially spirometry) facilities at the PHC level and definitely at District Hospital Level.

Equipping the primary health care level with required drugs and equipment, namely bronchodilators for long term management and inhaled bronchodilators, corticosteroids, antibiotics and oxygen for acute exacerbations.

Surveys to find out magnitude of the problem.

3. Tertiary Intervention

Establishment of referral centers at each district level.

Summary

Crucial tools in the battle against COPD are the (i) surveillance and analysis of the risk factors in a population; (ii) primary prevention to reduce the level of exposure to tobacco, poor nutrition, and environmental air pollution (indoor, outdoor, and occupational); and (iii) secondary and tertiary prevention by identifying cost-effective early screening and disease management interventions.

REFERENCES

British guideline on management of asthma. Revised June 2009

D.Wayne Taylor. *The Burden of Non Communicable Diseases in India.* Hamilton ON: The Cameron Institute 2010.

Expert Panel Report 3: *Guidelines for the Diagnosis and Management of Asthma*
Full Report 2007 National Institutes of Health

GINA. *Global strategy for asthma management and prevention* Updated 2009.

http://www.who.int/mediacentre/factsheets/fs307/en/index.html : Asthma. Accessed on 24.01.2012 at 1400hrs.

Nygren-Krug. H (2009). *A Human Rights-Based Approach to Non Communicable Diseases.* WHO. Geneva, Switzerland.

WHO (1995). *Countrywide Integrated Non-Communicable Diseases Intervention (CINDI) Programme.*

WHO Pakistan (2004). *National Action Plan For Prevention and Control of Non communicable Diseases and Health Promotion in Pakistan.* WHO Pakistan Office.

WHO (2002). *Non Communicable Diseases in South-East Asia.* WHO South-East Asia Office.

GINA. Global strategy for asthma management and prevention Updated 2009

British guideline on management of asthma. Revised June 2009

Expert Panel Report 3: *Guidelines for the Diagnosis and Management of Asthma*. Full Report 2007 National Institutes of Health

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