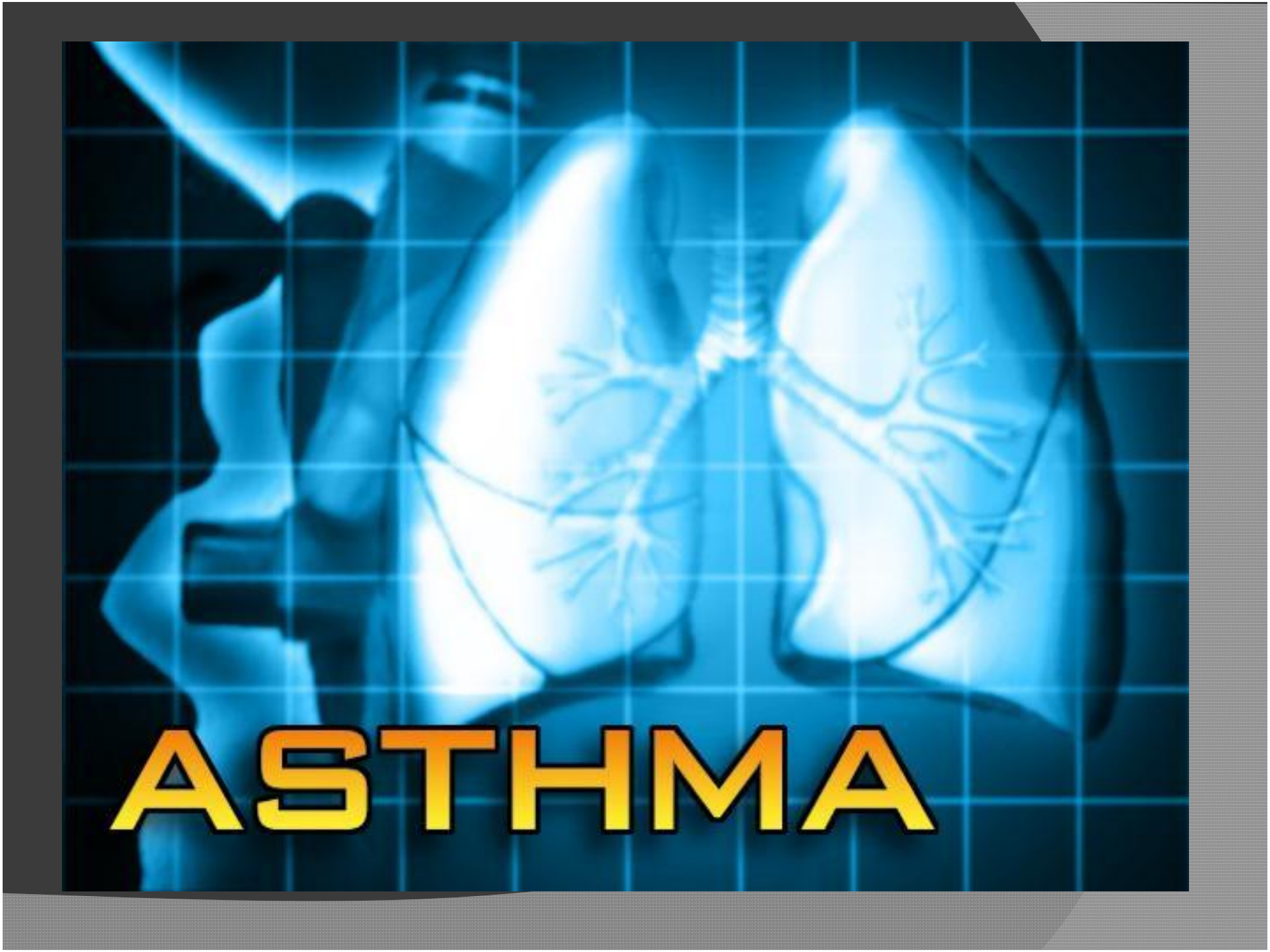


Definition:

characterised by chronic airway inflammation , increased airway hyper-responsineness, reversebile, leading to wheeze, cough, & dyspnea.

Are more common in boys, but following puberty females are more affected.

The socio-economic impact of asthma is enormous, when poor control lead to days lost from school or work, hospital admission & premature death.

Ateology:

may protect against asthma	may predispose to asthma
* living on farm	* childhood infection e.g. RSV
* large families	* allergens e.g. house dust mite
* childhood infection(parasite)	* indoor pollution.
* lactobacilli in gut flora	* deficiency of antioxidants
* exposure to pets in early life	* exposure to pets in early life.

Theories about ateology:

1- the hygiene hypothesis: decreased infections in early life bias the immune system towards an allergic phenotype. Increase infection in childhood e.g day care centers Th2 to Th1 to fight viral or bact infection.
In contrary RSV increase risk of asthma.

2- atopy: suggests that sensetization & exposure to allergens is important .
Warm, humid favour multiplication of house dust mites lead to asthma, in contrary early exposure to pet may protect against asthma(farmer).

3- nutritional theaory: milk fat & antioxidant as vit E & selenum may protect against asthma. In contrary early exposure to cow's milk lead to asthma.

4- obesity.

5- genetic (multifactorial).

Pathophysiology:

inhalation of an allergen rasult in two phase bronchoconstrictors:

type I reaction (early) lead to mediator release.

Type II reaction (late) inflamotery cell recruitment & activation.

The inhaled allergen interact with mucosal mast cell via an IgE immunogl leading to bronchoconstriction.

The cardinal pathophysiological features:

1- airway limitation: reversible.
2- airway hyper-reactivity: exaggerated bronchoconstriction to a wide range of stimuli e.g. excersize, cold air.
3- airway inflammation: infiltration of eosinophils, lymphocytes, mast cells associated with odema, smooth muscle hypertrophy & hyperplasia, thickening of basement membrane, mucus plugging & epith damage.
*chronic disease leading to fibrosis of airway wall & fixed narrowing of airways.

Risk factors:

1- allergy & allergens: animals(cats), house dust mites, indoor fungi.
2- drugs:
3- resp infection:
4- irritants: cigarette smoke , car exhaust.
5- chemicals: epoxy resins.
6- physical activity: exercise.
7- emotion.

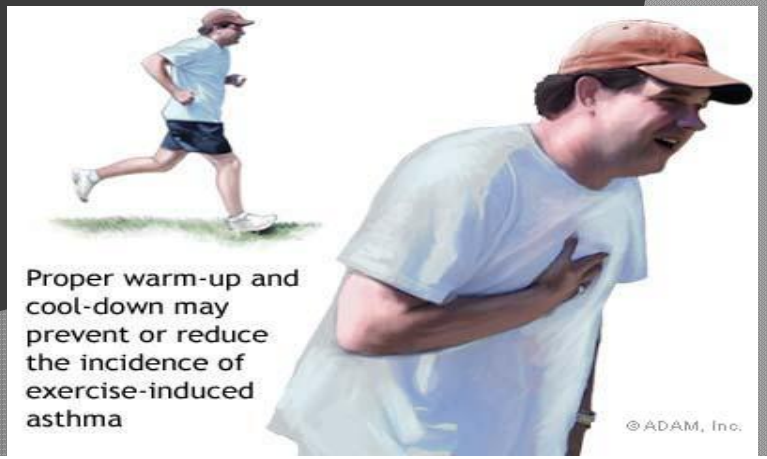
Types of asthma:

typeI	Type II
childhood	* adults
*atopic (extrinsic)	*non-atopic(intrinsic)
*+ve FH	* -ve FH
*+ve skin test	*-ve skin test
*T cells express IL 4&5	*T cells express gamma-interferon.

(occupational, exercise induced asthma, brittle asthma, cardiac asthma, nocturnal asthma, cough-variant asthma.

Clinical features:

*recurrent episodes of wheezing, dyspnea, & cough. & O/E
*mild intermittent asthma.
*persistent asthma: chronic form of wheeze & cough.
*severe acute asthma: its life threatening attacks in which the patient adopt an upright position, fixing the shoulder girdle to assisst the accessory muscle



Asthma Severity

Intermittent	**"Rules of Two"** <2x/week <2x/month at night
Persistent	Mild: <daily Moderate: daily Severe: continual

Adapted from "RULES OF TWO," a trademark of the Baylor Health Care System.

National Asthma Education and Prevention Program. *Expert Panel Report: Guidelines for the Diagnosis and Management of Asthma. Update on selected topics 2002.* Bethesda, Md: National Heart, Lung, and Blood Institute, National Institutes of Health, 2003. NIH publication 02-5074.

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()investigations:

1- spirometry: low FEV1 & VC with low FEV1/VC.
2- Peak flow meter: (morning dipping)
3- bronchial challenge test:
4- skin prick test:
5- CXR:
6- elevated sputum or peripheral blood eosinophil count.

To make a diagnosis of asthma need:

compatible clinical history plus either/or:

*FEV1 ≥ 15% increase following bronchodilator/trial of CS.
*> 20% diurnal variation on 3 days or more in a week on PEF.
*FEV1 ≥ 15% decrease after exercise.

Management:

A-general:

B- a stepwise approach:

step 1: occasional use of inhaled short-acting B2 agonist.

Step 2: adding inhaled CS

step 3: either increase the dose of inhalar CS or add long-acting B2 agonist or add lukotriene receptor antagonist.

Step 4: add high dose of inhaled CS & regular bronchodilators.

Step5: addition of oral CS

If the above symptoms non control, Rescue treatment is indicated:

its short courses of oral prednisone 30-60 mg daily , continue for 2 days after controlling symptoms, tapering not indicated:

* symptoms & PEF progressively worsen day by day.
* fall of PEF below 60% of patient's personal best recording.
* onset or worsening of sleep disturbance by asthma.
* persistence of morning symptoms until midday.
* progressively decrease response to an inhaled bronchodilators.
* symptoms severe enough to require Rx with nebulizer or inject able bronchodilators

()management of acute severe asthma:

firstly assess the patients:

features of acute severe asthma:

* PEF 33-50% predicted(<200 l/min)
* RR ≥ 25/min
* HR ≥ 110/min
* inability to complete sentences in 1 breath.

Life threatening features:

* PEF < 100L/min.
* O2 saturation < 92% or Pa O2 < 8Kpa
* normal PaCO2.
* silent chest.
* cyanosis.
* feeble resp effort
* bradycardia or arrhythmia.
* hypotension
* exhaustion
* confusion
* coma

near fatal asthma:

* raised PaCO2 &/or require mechanical ventilation.

So after assess the patient start treatment with:

* high O2.
* high dose of bronchodilators(salbutamol, salmetrol, ipratropium bromide)
* IV aminophylline
* IV magnesium
* IV fluids with K.
* IV leukotriene receptor antagonist
* mechanical ventilation indicated in:

coma, resp arrest, PaO2<8KPa & falling, PaCO2 > 6KPa & rising, PH low, exhaustion, confusion, drowsiness.

Discharge of patients:

* stop nebulizer therapy for at least 24 h
* PEF reach 75% of predicted
* ask the pt to avoid risk factors.

Does GERD trigger cough and asthma? Proposed mechanisms

- Microaspiration causing broncho-constriction (reflux theory)
- Receptors in esophagus causing bronchospasm *via* a vagal reflex (reflex theory – "gastric asthma")
- Receptors in esophagus causing an inflammatory process in the airways (neurogenic inflammatory model)

Tuchman et al, Gastroenterology 1984; 87: 872

Does asthma trigger GERD? Proposed mechanisms

- Coughing increases intra-abdominal pressure thereby increasing the pressure gradient across the LES
- Hyper-inflation alters the relationship between the crural diaphragm and the GE junction
- Airway obstruction causes negative intra-thoracic pressure
- Asthma medications lower LES pressure

() Chronic Obsetrictive Pulmonary Disease:(COPD)

()Definition:

is a chronic slowly progressive disorder, consisting of overlapping pathological processes including chronic bronchitis, chronic bronchiolitis & emphysema.
Chronic bronchitis: define as pt coughing up sputum on most of days of at least 3 consecutive months for more than 2 successive years.
Emphysema: pathological process of a permanent destructive enlargement of the airspaces distal to the terminal bronchioles.

() Risk factors:

* smoking *biomass solid fuel fires *occupation(coal & gold miners)
*outdoor & indoor pollution *low socioeconomic status.
*low birth wt *infection viral *cannabis smoking
*decrease in vit C & E *genetic (alpha 1 antiproteinase deficiency)

() Path physiology:

pulmonary:

* enlargement of mucus secreting glands & increase in no. of goblet cells
* pul vascular remodeling & impaired heart function (cor pulmonale)
* loss of elastic tissue, gas trapping & chest hyperinflation
* unopposed action of proteases & oxidants leading to destruction of alveoli

systemic:

* muscular weakness reflecting deconditioning & cellular changes in skele mus
* increased blood viscosity
* impaired salt & water retention
* altered fat metabolism leading to wt loss
* increase prevalence of osteoporosis

() Clinical features:

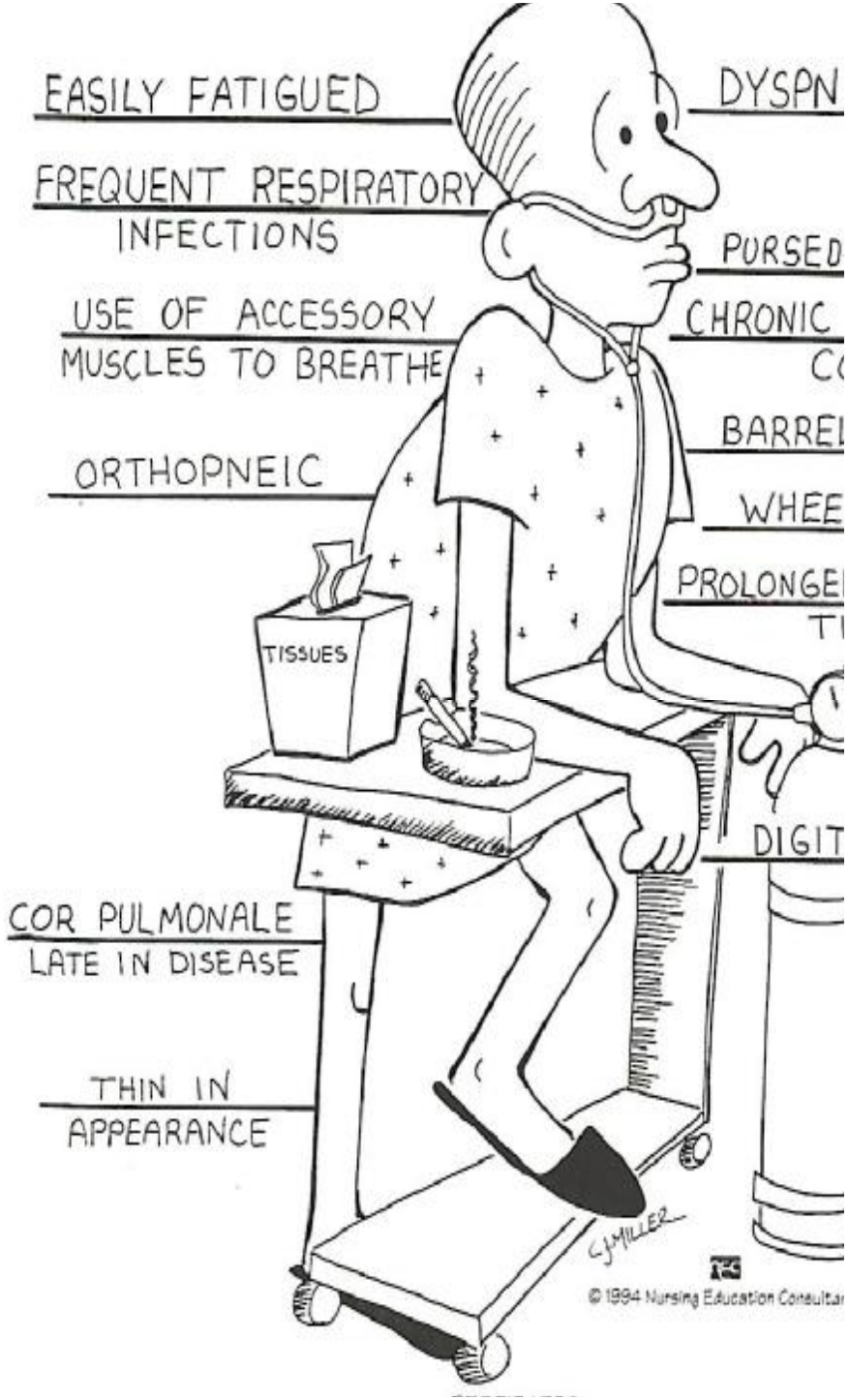
* persistent cough & sputum &/or dyspnea.
* hemoptysis & clubbing of fingers.
* symptoms:

()obstructive:

* use of accessory muscle
* excavation of suprasternal & supraclavicular fossae with in drawing of intercostal spaces.
* decrease cricosternal distance
* tracheal tug
* increased A-P diameter to lateral diameter
* pursed lip
* wheeze

()CO2 retention:

* warm hand & sweaty
* bounding pulse
* flapping tremor
* central cyanosis
* papillodema

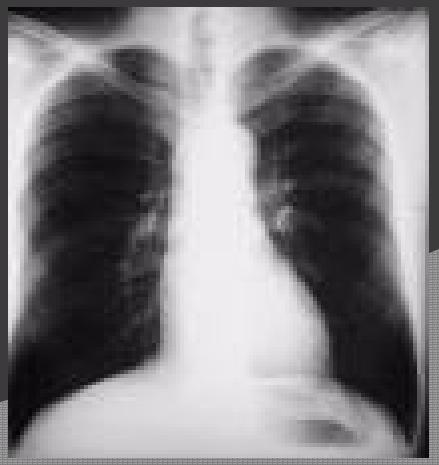


()cor-pulmonale:
*peripheral edema
*raised JVP
*RV heave, loud p2, TR
*tender hepatomeagally

()Investigations:

1- CPC
2- alpha-1 antiprotienase level
3- CXR normal, hypertranslusent lung, low flat diaphragm, small size heart, bullae
4- PFT low FEV1 , VC , low FEV1/VC, low PEF , increased lung volumes,
5- CT scan
6- pulse oximetry
7- blood gas analysis.

()Classification: according to FEV1
*mild : 50-80%
*mod:30-49%
*sever: <30%



() Management:

1- general: stop smoking & avoid dusty environment
2- bronchodilators
3- CS whether oral or inhalar
4- pul rehabilitation
5- O2. long-term domiciliary O2 therapy LTOT it improve survival, prevent pul hypertension, decrease secondary polycuthemia,
low conc with minimum 15 h per day, indicated in:
a-PaO2 < 7.3 kpa irrespective of PaCO2
b- PaO2 7.3-8 kpa plus pulm HPT , periph odema, or nocturnal hypoxemia.
C- stop smoking
6- surgery:
a- young pt with large bullae
b- lung reduction surgery
7- others:
()annual infleunza, & 3-5 yearly pneumococcal vaccinations
()improve obesity, poor nutrition, depression & social isolation
()mucolytic therapy.