Asthma

Group 4: Arsh, Charankamljit, Devon, Janna, Jas, Mandeep (Mandy), Monika, Sarah & Trixia
What is Asthma?

- A chronic inflammatory disorder of the airways, which can cause different degrees of obstruction= wheezing, breathlessness, chest tightness & coughing (particularly at night & early morning)
- Occurs as a result of environmental effects on the airway, these effects can be endogenous and exogenous
- The environmental effects trigger an immune response which leads to airway inflammation & bronchoconstriction
Triggers of Asthma Attacks

- When the airway becomes more inflamed, it also becomes more hyper-responsive, but the exact mechanism for why that happens is unknown.
- Involves multiple stimuli and triggers.
- Some triggers include:
  - Allergens
  - Exercise
  - Respiratory Infection
  - Nose/Sinus Problems
  - Drugs/Food Additives
  - Air Pollutants
  - Gastroesophageal Reflux Disease
  - Emotional Stress
Triggers Continued

- **Allergens:** have an exaggerated immunoglobulin response (ex: dust, pollen, grasses, mites, roaches, moulds, animal dander, latex) the mast cell complexes remain for a while so when there is a second exposure mast cells are triggered for degranulation.

- **Exercise:** during physical exertion, typically occurs after activity with cold or dry air & is characterized by bronchospasm that causes cough, SOB, wheezing, chest tightness or a combo of these.

- **Respiratory Infection:** cause increased inflammation in tracheobronchial system= increased airway hyper-responsiveness, which can last 2-8 weeks.

- **Nose/Sinus Problems:** allergic rhinitis, seasonal/perennial, and nasal polyps, usually related to inflammation of the mucous membrane; bacterial sinusitis might occur.
Triggers Continued

- **Drugs/Food Additives**: People with asthma have *asthma triad*: nasal polyps, asthma, sensitivity to aspirin & NSAIDs. Wheezing can develop after 2hrs after taking them; salicylates cause a reaction as well. B-adrenergic blockers in oral form, topical eye drop trigger bronchospasm & ACE inhibitors can induce cough & worsen asthma symptoms. Tartrazine and sulphites found in food can cause asthma symptoms.

- **Air Pollutants**: cigarette or wood smoke, vehicle exhaust, diesel particulate, elevated ozone levels, sulphur dioxide & nitrogen dioxide trigger asthma attacks; heavy industrialized areas, climate conditions can cause heavy concentration pollution in the atmosphere

- **Gastroesophageal Reflux Disease**: mechanism unknown; reflux of stomach acid into the esophagus aspirated into the lungs = reflux vagal stimulation & bronchoconstriction. GERD can trigger daytime asthma as well.

- **Emotional Stress**: psychological stress with extreme emotion (crying, laughing, anger, fear) can lead to hyperventilation & hypocapnia= airway narrowing
Pathophysiology

- Exposure to allergens starts an inflammatory response that involves different types of cells, mediators & chemokines
- Two types of responses:
  - Early-Phase Response
  - Late-Phase Response
- Pathophysiological features: reduction in airway diameter & increase in airway resistance due to mucosal inflammation, constriction of bronchial smooth muscles & excess production of mucus
- Also causes: hypertrophy of bronchial smooth muscle, thickening of basement membrane, hypertrophy of mucous glands, secretion of thick sputum, hyperinflation & air trapping in alveoli = increase work of breathing
- Due to the events above can cause altered respiratory muscle function, altered arterial blood gas (ABG) levels, and abnormal distribution of ventilation & perfusion
- If it is not treated is can cause irreversible lung damage
Pathology of Asthma

- **Normal airway**: Relaxed smooth muscles
- **Asthmatic airway**: Wall inflamed and thicken
- **Asthmatic airway during attack**: Air trapped in alveoli, tightened smooth muscles
Early-Phase Response

- Bronchospasm = increased mucus secretion, edema formation & increased tenacious sputum (wheeze, cough, SOB)
- Triggered when allergens connect to receptors on mast cells under the basement membrane of the bronchial wall
- Mast cells release granules & disrupt the cell membrane = release inflammatory mediators (histamine, bradykinin, leukotrienes, prostaglandins, platelet-activating factor, chemotactic factors & cytokines)
- Exercise can cause a similar response; mediators cause intense inflammation, bronchial smooth muscle constriction, epithelial damage & increased vasodilation/permeability
- Peaks within 30-60 minutes after exposure and subsides after 30-90 minutes
Late-Phase Response

- Can be a more severe response
- Primary characteristic is inflammation
- Eosinophils & neutrophils infiltrate the airways and release mediators that cause more inflammation & cause mast cells to degranulate so histamine is released & other mediators
- Increases airway reactivity which can reduce the threshold for exposure for future attacks
- Peaks 5-12 hours after exposure and can last from a couple of hours to a couple of days
Diagram of the Pathophysiology of Asthma (pg. 711)
How Does Asthma Work?

The following video explains how asthma works!

https://www.youtube.com/watch?v=PzfLDi-sL3w
How To Use Different Types of Inhalers

https://www.youtube.com/watch?v=5xrIBFRufmQ
The preferred method of medication delivery for asthma is by inhalation, which enables the direct deposition of medication to the affected airways, leading to better efficiency and fewer side effects. While there are a number of different devices available, the most used is the pressurized MDI, which requires proper technique:

1. Shake the MDI
2. Breath out to the end of a normal breath;
3. Actuate through an open mouth, just after inhalation begins;
4. Breath in slowly over 4 s to 6 s, to full lung volume;
5. Hold breath 5 s to 10 s; and
6. Wait 30 s to 60 s, shake and repeat above steps.
Asthma Management

- Very mild, intermittent asthma may be treated with fast-acting beta2-agonists taken as needed
- Inhaled corticosteroids (ICS) should be introduced early as the initial maintenance treatment for asthma
- Leukotriene receptor antagonists (LTRAs) are second-line monotherapy for mild asthma
- If asthma is not adequately controlled by low doses of ICS, additional therapy should be considered
- In children six to 11 years of age, the ICS should be increased to a moderate dose before an additional agent such as a long-acting beta2-agonist (LABA) or LTRA is added
- In children 12 years of age and over, and adults, a LABA should be considered first as add-on therapy only in combination with an ICS
Asthma Management Continued

- Increasing to a moderate dose of ICS or addition of a LTRA are third-line therapeutic options. Theophylline may be considered as a fourth-line agent in adults.
- Severely uncontrolled asthma may require additional treatment with prednisone.
- Asthma symptom control and lung function tests, inhaler technique, adherence to asthma treatment, exposure to asthma triggers in the environment and the presence of comorbidities should be reassessed at each visit and before altering the maintenance therapy.
Asthma Management Continuum
Children (6 years and over) and Adults

Regularly Reassess
- Control
- Spirometry or PEF
- Inhaler technique
- Adherence
- Triggers
- Comorbidities

Adjust Therapy to Achieve and Maintain Control

Inhaled Corticosteroid (ICS)*
*Second-line: Leukotriene Receptor Antagonist (LTRA)

Low Dose
≥12 yrs: ≤250 mcg/day†
6-11 yrs: ≤200 mcg/day†

Medium Dose
251 – 500 mcg/day†
201 – 400 mcg/day†

High Dose
>500 mcg/day†
>400 mcg/day†

Fast-acting Bronchodilator on Demand

Environmental Control, Education and Written Action Plan

Confirm Diagnosis

Controlled

Uncontrolled

†HFA Beclomethasone or equivalent; *Second-line: LTRA; †Approved for 12 years and over.
References


