

Drugs used in Anaphylaxis

Objectives:

- ◆ Perceive the differences between anaphylactic shock and other types of shock
- ◆ Recognize its nature, causes & characteristics.
- ◆ Specify its diagnostic features
- ◆ Identify its standard emergency management protocol
- ◆ Justify the mechanism of action and method of administration of each of the different used drugs to limit its morbid outcomes



ANAPHYLAXIS :

Is a sudden, severe allergic reaction affecting the whole body.

The severe allergic symptoms including:

- Rash
- Mucosal swelling
- Difficulty breathing
- Reduced blood pressure → the blood cannot filtrate the contents of the tissues



ANAPHYLACTIC SHOCK

A life-threatening allergic reaction that causes shock (hypoperfusion) and airway swelling



Means that the tissues cannot take enough nitration and oxygen from the blood



It can cause ischemia, fibrosis or death

Generalized circulatory derangement causing multiple organ HYPOPERFUSION [Inadequate oxygen delivery to meet metabolic demands] & strong sympathetic activation

Types of shuck

Types of shuck	Examples
Hypovolemic shuck	Haemorrhage (loss of blood ex: by accident) or fluid loss (plasma, ECF)
Cardiogenic shuck	Inability to contract & pump → myocardial infarction
Obstructive shuck	Extracardiac obstruction → Pul. embolism, cardiac tamponade
Distributive shuck	↓ PR → septic shock, neurogenic, anaphylactic shock (↓ blood in the circulation and ↑ in tissues)

ANAPHYLACTIC SHOCK THERAPY PROTOCOL

1st Line

1-Adrenaline:

Mechanism:

A nonselective AD agonist

[α_1 , α_2 , β_1 , β_2]

Actions:

As an α -AD agonist

-Reverses peripheral vasodilation → maintains BP & directs blood flow to major organs.

-↓ edema → reverse hives, swelling around face & lips & angioedema in nasopharynx & larynx.

As a β -AD agonist

-Dilates bronchial airways +↓ histamine & leukotriene release from mast cells → β_2 effect.

-↑ force of myocardial contraction → β_1 effect

Contraindications:

Rare in a setting of anaphylaxis

Not given > 40 y cardiac patient

ADRs:

(Dysrhythmias) which means abnormal electrical activity in the heart.

Administration

Best is (IM) route in anaphylaxis. Why?

- Easily accessible
- Greater margin of safety → no dysrhythmias as with IV
- No need to wait for IV line → if present
- given by physician under monitoring.

Repeat every 5-10 min as needed

Patients observed for 4-6 hours. Why?

Fear of biphasic anaphylaxis.

-If hypotension persist → start dopamine

2nd Line

1-CORTICOSTEROIDS:

Not used alone → not life saving.
Given slowly IV or IM.

Actions:

- Reverse hypotension & bronchoconstriction →
- ↓ release of inflammatory mediators (anti-chemotactic & mast cell stabilizing effects).
- Decrease mucosal swelling and skin reaction.
- May help to limit biphasic reactions →
- ↓ allergic mediators.

It has two actions

genomic action
that takes hrs –dys
and bind to
Cytosolic receptors

Non-genomic action
rapid and
acting on Membrane bound receptors
modulating 2nd messengers levels →
(within minutes)

2-H₁ BLOCKERS:

- It **cannot** be used alone → not life saving
- Given slowly intravenously or intramuscularly.
- May help to **limit** biphasic reactions by ↓ more histamine release.

3-H₂ BLOCKERS:

- Block the effects of released histamine at H₂ receptors.
- Ramifying the heart & some BV
- Responsible for glandular hypersecretion.
- They are given in adjuvance to H₁ blockers → additive benefit over H₁ blockers alone in treating anaphylaxis.

Adjuvant to 2nd line

1-Bronchodilators :

Inhalational

Salbutamol:

- β_2 -AD agonist
- short acting, rapid relief onset
- relax bronchial smooth muscle and may decrease mediator release from mast cells and basophils.
- It may also inhibit airway microvascular leakage.

Ipratropium:

- Anticholinergic
- longer duration of action → ↓ secretion.

Parental

Aminophylline IV:

- may be useful in the treatment of anaphylaxis when inhaled bronchodilators are not effective & bronchospasm is persistent.

- Given in hospital setting as levels of drug should be Therapeutically Monitored → has narrow therapeutic index

2-Glucagon:

- Drug of choice for severe anaphylaxis in patients taking β -blockers
- Has both positive inotropic & chronotropic effects on heart → ↑ cardiac cyclic AMP → an effect entirely independent of AR
- That is why effective in spite of beta-adrenergic blockade.
- Efficacy of acting on bronchi < heart → **no evident bronchodilation**