3rd pharmacology lecture "Anaphylactic Shock"

Objectives:

- Define the anaphylactic shock, its causes and drugs used in the treatment.

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The first 7 points are for understanding only not for memorizing.

1) Shock: sever drop in blood presser + difficulty in breathing (not always but in anaphylactic shock)
What is the difference between shock and hypotension?
Both are: decreasing in blood presser
In Shock ONLY: hypoperfusion
2) Hypoperfusion: the blood has no ability to reach the peripheral circulation in appropriate amount to provide O_2 and to take CO_2.
The tissues can't take enough nitration and oxygen from the blood. It can cause ischemia, fibrosis or death
3) Anaphylaxes shock: A life-threatening allergic reaction that causes shock (Hypoperfusion) and airway swelling.
Is a sudden severe allergic reaction affecting the whole body instead of one or two areas.
More in younger age cause the more we grow allergic reactions decrease.
The severe allergic symptoms including:

- ✓ Rash
- ✓ Mucosal swelling
- ✓ Difficulty breathing
- ✓ Reduced blood pressure→the blood can't filtrate the contents of the tissues.

4) During shock there is a sympathetic over activity.
During shock, there is a severe drop in blood presser, as a normal reaction from my body the sympathetic system will be over active to return the blood presser to its normal level.
Failure in sympathetic causes shock, which could cause death if the patient didn't get a quick treatment.

5) Distributive shock: peripheral resistance for blood flow => hypoperfusion => shock:
1) Comes from allergic reaction by antigen-antibody reaction (hyper sensitivity type1) shock (the common type)
Primary and secondary exposure:
Primary: antigen recognition
Secondary: antigen-antibody reaction => digranulation of mast cell =>different types & levels of allergic reactions, start from skin involving, cause shock.

2) Another type of shock, when it's allergic but without antigen-antibody reaction condition => anaphylactoid shock

Examples:
A patient came to X-ray department, after they injected him a specific kind of colorizing material he got into a sever shock (hyper sensitivity type1)

A doctor gave his patient certain drugs (morphine or muscle relaxants) that beat Histamin, the patient had a shock (without antigen-antibody reaction => not allergic=> anaphylactoid shock)

Dangerous of shock related to:
1) Severe dyspnea & cardiovascular collapse
2) Edema of larynx

6) Rescue therapy:
Once diagnose is made that the condition is shock:
1_Call SOS
2_Insure two things: respiratory support + circulatory support
In developed countries, patient already has his Kit (specialized therapy) If he's already collapsed, turn him upside down before injecting.

Kit: adrenaline, which has:
α effect: increase BP in peripheral vessels.
β 1 effect: increase heart pulses.
β 2 effect: bronchodilatation + blood vessels to the heart vasodilator (coronary & muscular blood vessels dilatation)

7) Biphasic reaction: the drug has a short time effect.
The recurrence of symptoms within 1–72 hours with no further exposure to the allergen.

Pharmalogical part:

Adrenaline: non selective adrenergic agonist
Mechanism of action in anaphylaxis:
α: Increase BP, decrease edema.
β (general): Dilatation of bronchi, increase force of muscle heart contraction (+ supplying O_2 by β 2 to the heart)

* Adrenaline increases sympathetic activity => in conditions of Hypertension, myocardial infarction or heart failure it might cause more problems to the heart especially for older people, but we can use it in hospital cause the patient is already under observation.

*It has a biphasic reaction.
* It's given intra venues drop by drop (in hospital). But if the patient is in dangerous & far away from the hospital it could be given intra muscular.
* Twice per 5-10 minutes if it's given intra muscular.
* In patients use adrenergic blockers drugs, Adrenaline will have a very low effect.
People who use β blockers Adrenaline are useless => scroll down the solution is written in pink.

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Corticosteroid: control immune reactions
In anaphylactic shock:
*It's NOT life saving (2nd line).
*Reverses hypotension and bronchoconstriction.
*Reverses anaphylactic shock.
*Decrease mucosal swelling, skin reactions and edema.
*Slow action on nuclear receptors (genomic action) but rapid action on membranous receptors (non-genomic action).
*Decrease biphasic reaction: long time effect (as long as it works on nuclear receptors).
*Given in hospital
*Given slowly IV or IM.

Anti-histaminic (H1 blockers):
*Decrease histamine.
*Some of the drugs decrease another allergic mediators are called: anti histaminic with anti allergic actions.
*Very weak drugs in cases of anaphylaxes and asthma cause they only block histamine receptors, while other mediators have the ability to block it & to mediate anaphylaxes at the same time.
*NOT life saving at all.

Bronchodilators:

1) Adrenaline: already taken

2) Salbutamol:

*Acts on β 2

*Helpful in asthma & anaphylactic shock

* Life saving.

*Rapid relief onset.

3) Ipratropium:

*Acts on parasympathatic system.

*used in sustained asthma.

*BUT SLOW reacting on anaphylactic shock.

4) Aminophylline:

*Efficient bronchodilator.

*Has a toxic effect.

*Given in hospital.

*Increase contraction of heart and cAMP.

1st line: Adrenaline _ 90% except patients use an adrenergic blockers or cardiac patients.
2nd line: corticosteroid
3rd: salbutamol

In patients use β blockers: aminophylline + glucagon.

If β receptors are blocked:

n order to dilate patient bronchi we can use: aminophylline
Which works in a very different way not by β receptors.

While to increase heart contraction we might use: Glucagon
Glucagon + β 1 receptors are increasing cAMP