

ARDS/SEPTIC SHOCK/DIC/MODS/BLOOD TRANSFUSION

ARDS Acute Respiratory Distress Syndrome

Causes-	Trauma	Burns	Drugs
	Severe Infection	Sepsis	Open heart surgery
	Fat Emboli	Aspiration	Fluid overload
	Prolong Ventilation		

Main issue is pulmonary edema and the affect of the edema on the alveoli. Damage may occur to the pulmonary capillaries by many different methods, such as: a direct method drugs or ventilation, or an indirect method, inflammation or DIC. Capillary damage leads to pulmonary edema with all of its' complication.

Pulmonary Edema

Pulmonary Hemorrhage

Decreased Gas Exchange

Vasoconstriction

Reduced Compliance of Blood Vessels

Pulmonary Hypertension

Reduced Lung Volume

Decrease in Surfactant

General Hypoxia

Collapse of Alveoli

Clinical Signs:

Shallow rapid breathing, with no history of Cardiac Disease

Restlessness or anxiety due to general hypoxia

Tachycardia due to general hypoxia

Initial PCO₂ will be normal or low because the CO₂ diffuses out of the body better than the O₂ but it will rise in the late stages leading to acidosis

Pain on inspiration

Hyper-resonance upon chest percussion due to edema

Cerebral hypoxia may lead to confusion

General treatment

Treat the underlying cause

Fluid restriction

Position high Flowers

Diuretics / corticosteroids

Septic Shock

Cause: Invasion of a microorganism mainly gram negative but some maybe gram positive, as in the case of Toxic shock, also maybe the result of a fungi or a virus.

Microorganism **release of toxin** **inflammatory response**

Remember the key to inflammation is vasodilatation

Maldistribution:

Some areas of the body will be hyperperfused, while other areas will be underperfused

Massive peripheral vasodilatation- pink warm flushed skin

Increased intravascular fluid shift to the third spaces leads to increase in blood viscosity (increased Hematocrit)

Microemboli formation leads to decrease tissue perfusion

Signs

Increased temperature due to pyrogens

Increased metabolic rate

Increased Heart rate (bounding pulse)

Pulmonary edema

Decreased Urine output

Metabolic acidosis from the build up of lactic acid

DIC Disseminated Intravascular Coagulation

Intense clotting followed by intense clot lyses leading to the depletion of clotting factors. The intrinsic clotting maybe started by endotoxins, which rough up the inside of the small vessel, or by the low flow rate which cause platelets to rupture. The organs most affected are the lungs, kidneys, brain and skin

Causes

Sepsis

Anaphylactic shock

Trauma

Malignancies

Lupus

Sickle Cell Crisis

Burns

Transfusions

OB Causes:

Eclampsia

Septic abortion

Abruptio placenta

Signs

Bleeding

Petechiae and bruising

Bleeding from several areas- IV, mouth, and GI system

Cerebral bleeds leads to confusion

Hematuria

Bleeding in the lungs and coughing up blood

Clotting

Kidney failure
CNS involvement may have appearance of a CVA or stroke

Labs

Increase in PT, PTT and Thrombin time

Multiple Organ Dysfunction Syndrome MODS

Failure of 2 or more organ systems after a severe illness or injury.
Mortality Rate of 60%- 90%

Causes:

Major surgery or trauma ARDS Burns Necrotic tissue
Acute pancreatitis or renal failure Sepsis

Clinical manifestations are based on inflammation, tissue hypoxia, and hypermetabolism

Days 1-7

Altered mental status
Respiratory involvement or respiratory failure
Hyperdynamics
 Increased temperature due to pyrogenics
 Increased Heart rate (bounding pulse)
 Pulmonary edema
 Decreased vascular resistance
 Hypotension
Hypermetabolic
 Increased metabolic rate
 Depletion of O₂ and stored fuel (ATP)

Days 7-14

Hyperdynamics and Hypermetabolic states intensify
Bacteremia- presence of bacteria in the circulating blood
Signs of impending liver and kidney failure
Respiratory failure

Days 14-24

Liver and kidney failure
GI and the immune system fail
Respiratory failure progresses to ARDS

Days 24-28

Death

Treatment

If the initial insult is known aggressively treat the source
Restore and maintain oxygenation
Provide nutritional support for teaching

BLOOD TRANSFUSION

BLOOD TYPE	ANTIGEN ON THE RBC	ANTIBODY IN THE PLASMA
O	NONE	ANTI-A ANTI-B
A	A	ANTI-B
B	B	ANTI-A
AB	BOTH A&B	NONE
Rh+	Rh	NONE
Rh-	NONE	POSSIBLE ANTI-Rh

Antigens are markers on cells, they are the way in which the body recognizes its' own cells vs. foreign cell and pathogens. Antibodies are proteins in the blood plasma which destroy antigens: each antibody is specific to a certain antigen. Example Anti-B antibody will clump and destroy the antigen B on a foreign blood cell, just like the chicken pox antibody will only attack the chicken pox virus.

Types of transfusions:

Packed RBCs- used to increase oxygenation of a patient. Examples: Hypovolemic shock or anemia. This transfusion contains few plasma elements.

Fresh Frozen Plasma- includes many clotting factors used to aid in cases of severe bleeding, liver failure, or too many blood thinners.

Platelets- aids in blood clotting may be used with patients having thrombocytopenia

Albumin- the major plasma protein which determine the oncotic pressure of the blood, when administered it will pull fluid back into the blood. Used in cases of liver disorders and severe burns.

Types of reactions

Immune:

Hemolytic RBC antigen / antibody incompatibility

Caused mainly by mismatched blood, often happens within the first 15 minutes.

Symptoms include: restlessness, anxiety, flushing, chills, headache, nausea, chest pain due to heart involvement, loin pain due to kidney involvement, tachypnea, tachycardia, hemolytic shock, DIC, kidney failure and death.

Blood sample will show a rise in hemoglobin as the RBCs begin to lyses

Febrile Reaction:

Incompatibility of the WBCs, platelets or Igs, usually happens in the first 30 minutes
Symptoms include: increase in temperature (fever) chills and headache

Non-Immune:

Circulatory overload: fluid volume overload

May occur up to 24 hrs. after the transfusion, common in patients with previous cardiac or renal problems.

Symptoms include: Pulmonary edema (cough, crackles...etc.) Increase in pulmonary capillary wedge pressure

Massive Transfusion: causes thrombocytopenia (dilution of platelets)

Symptoms include: ecchymoses, purpura and bleeding

Hypothermia may also be a side effect of a massive transfusion

Also watch for electrolyte imbalances

Transmission of infections

Examples: HIV, hepatitis B&C, syphilis, malaria, toxoplasmosis, and CMV

Intoxication: caused by the build-up toxins in the stored blood

Potassium: from the breakdown of cells in the blood

Ammonia: from the breakdown of cells in the blood

Citrate: from an anticoagulant mixed with the blood, often seen in a transfusion using more than 4 units blood. This reaction may also lead to hypocalcemia as the Ca⁺ binds to the Citrate

Types of Donations:

Homologous: From a volunteer donor, think blood bank

Autologous: Patients bank their blood before a procedure, think self to self

Directed Donation: A specific donor, usually known to the patient, will donate to a specific patient, think friend to friend

Autotransfusion / Blood salvage: The patient's own blood which is lost during a trauma is cleansed and reused within six hours, think trauma self to self