

Subject :

Lec mo 8 Lecture 20 (Part 1 + 2)

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Hemodynamics 6 lectures

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Disseminated Intravascular Coagulation (DIC)



ملك بالله خطيرة ويتنتشريح لمحسم المين علمات + التزيي DIC is a thrombo-hemorrhagic disorder, characterized by systemic activation of the coagulation cascade by various stimuli , with hundreds of thrombi occluding microcirculation leading to hypoxia and microinfarcts (Serious dieases)

It is also called <u>consumptive coagulopathy</u>, followed by bleeding due to <u>consumption of platelets & clotting factors in</u> blood مود بسب تنزیف لابه عوامل ایتخش تم استملا کما للکوین <u>identification</u> (مالکان الکوان) Fibrin Hhrom bi نویس Therm of DIC;

- 1. Wide-spread endothelial cell damage
- 2. The release of tissue factor or thromboplastic substances into the circulation

(systemic)

- It is characterized by a sudden or gradual onset of widespread fibrin thrombi in the microcirculation.
- DIC is not a primary disease but rather is a potential complication of any condition associated with widespread activation of thrombin
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في لمبولس لازرق) ولمي ممكن يحفذوا fhrombi ممكن لمصناعفات تؤدي إلى Dic لتعقق العلاقة + الاعصاء لإكثر عرصة

• The major causes of which including <u>obstetric</u> <u>complications, infections, neoplasms</u>, <u>massive tissue injury & others</u>.

- Thrombin generation in DIC is initiated through the tissue factor/factor VII(a) pathway that activates downstream coagulation factors. (From extrensic Pathway)
- Tissue factor may be expressed by activated monocytes, but also by vascular endothelial cells or cancer cells.
- Histologic studies in patients with DIC show the presence of ischemia and necrosis due to fibrin deposition in small and medium-sized vessels of various organs.
- The presence of these intravascular thrombi appears to be clearly and specifically related to the clinical dysfunction of the organ.





Disseminated Intravascular Coagulation (DIC)



Manifestation of DIC ستع ليسية In the skin &- wide Spread Petchiae , ecchymo Sis

- In kidneys, microthrombi can result in numerous microinfarcts in renal cortex leading to bilateral renal cortical necrosis, then renal failure
- In brain, microthrombi & numerous micro infarcts in the brain
- Lungs and GIT involvement by microinfarcts
- The adrenals involvement leading to extensive bilateral adrenal hemorrhage called (Waterhouse Friedrichsen Syndrome) a group of symptoms caused when the adrenal alands fail to function normally

Disseminated Intravascular Coagulation (DIC)



Laboratory tests reveal:

- Thrombocytopenia

- Prolonged prothrombin time (PT) & partial thromboplastin time (PTT) (Fibronlysis)

Increase Fibrin degradation products (FDPs) (↑ D- Dimer)

Treatment: heparin & fresh frozen plasma, and treat the underlying





Figure 42 : Gross appearance of kidney showing renal cortical necrosis in DIC. In the adrenal May cause Chemorrgic adrenal Syn drome Called (Water house Friedri chsen syndrome)



Figure 43 : DIC in kidney : Microscopic view .





Figure 44 - Microscopic view of renal microthrombi in DIC.



Figure 45 : Gross appearance of lung showing features of DIC , numerous hemorrhagic microinfarcts & hemorrhages .



Figure 46 : Skin in DIC,



Embolism



Embolism



- An embolus is a detached intravascular solid, liquid, or gaseous mass that is carried by the blood to a site distant from its point of origin.
- 99% of all emboli represent some part of a dislodged thrombus, hence the term thromboenbolism. The most common

- Two forms: I.Pulmonary thromboembolism leads to hypoxia and right-sided heart failure.

Embolism

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Rare forms:

Air embolism, fat embolism, amniotic fluid embolism.



Thrombophlebitis is swelling (inflammation) of a vein

Phlebothrombosis is the presence of a clot within a vein, unassociated with inflammation of the wall of the vein

like deep Pain of the leg

Pulmonary Thromboembolism

• In 95% of cases, emboli originate from thrombi within deep leg veins, above the knee (DVT).

 $\begin{array}{ccc} \text{Deep Vein} & \longrightarrow & \text{Heart} & \longrightarrow & \text{lungs} \\ & & & & \\ & & & \\ & &$

• They are carried through progressively larger channels and pass through the right side of the heart to the pulmonary vasculature.



Pulmonary Embolism



لو سکوت کل لمنطقة دح معمل (Pulomnry Infraction)



Saddle Pulmonary Embolism







Thrombophlebitis versus phlebo (edema عربية مع thrombosis

- Thrombophlebitis is a condition in which inflammation of the vein wall has preceded the formation of a thrombus (blood clot).
- Phlebothrombosis is the presence of a clot within a vein, unassociated with inflammation of the wall of the vein ومانبتعالج

Effects of Pulmonary Thromboembolism :

- (1) Fatal, Sudden death, acute right heart (ventricular) failure, also called acute cor pulmonale <u>, occur when 60% or more of the</u> pulmonary circulation is obstructed with emboli.
- (2) Embolic obstruction of medium- sized arteries may result in:
 Pulmonary hemorrhage : but usually does not causes
 pulmonary infarction (in normal person) because of blood flow
 into the area from an intact bronchial circulation (normally
 there is double pulmonary blood supply from pulmonary &
 bronchial arterial circulations), however,
 Set of the state of t

Z)

(B) A similar embolus in the setting of left-heart failure (& resultant sluggish bronchial artery blood flow) may result in a large pulmonary infarction

ا في حال كان عنده فش في الجمة السرى (لتيحبة وكود لدم) -> Infraction



- (3) Embolic obstruction of small end-arteriolar pulmonary branches usually does not result in associated infarction.
- (4) Multiple emboli over time may cause pulmonary hypertension with chronic right heart failure (cor pulmonale).
- (5) Majority (60% to 80%) of pulmonary emboli are clinically silent because they are small. With time, they undergo organization & become incorporated into the vascular wall may undergo fibrosis leading to pulmonary hypertension



Cause Pulmonry trunk

F 47 : Fatal pulmonary thrombo-embolism (PTE). A large coiled-up thromboembolus . It lies within the Rt.V. outflow tract, filling the pulmonary trunk & the bifurcation of both Rt & Lt pulmonary arteries (saddle embolus).

6.31 Pulmonary embolism



D Elsevier, Kumar et al: Robbins Basic Pathology 8e - www.studentconsult.com

F 48 : Pulmonary Thrombo Embolism: Saddle embolus



F 49 : Recurrent pulmonary Thromboembolism (PTE). The secondary branches of a pulmonary artery have been opened to reveal two small emboli wedged within the vessels. Both have tapering distal extensions.



6.34 Recurrent pulmonary embolism

Systemic Thromboembolism

20%. Rallon Blug

- 80% arise from intra cardiac thrombi.
- The remainder (20%) originate from **aortic aneurysms** and thrombi overlying ulcerated atherosclerotic plaques. or from fragmentation of a valvular vegetation (of infective endocarditis);
 - only very rarely due to paradoxical emboli (emboli passing from the right heart through atrial or ventricular septal defect into the left heart & then in the aorta)

Effects:

ما بشبه الحالة الاولى الي حكينا عنها كان يتركز في مكان واحد هاي الحالة منتشرة لاكثر من مكان

- In contrast to venous emboli, which tend to lodge primarily in one vascular bed only (the lung in systemic venous circulation & the liver in the portal circulation)
- arterial emboli can travel to a wide variety of sites; the site of arrest depends on the point of origin of the thromboembolism





Major sites for arterial embolization are

- (1) Lower extremities (75%),
- (2) Brain (10%), with the intestines, kidneys, & spleen involved to a lesser extent.
- The consequences of systemic emboli depend on the: (collateral) الكن لعبةد على وجود (Infraction) يادة يحيس المعادية من المعالية على المعادين المعالية المعال
- (1) Collateral's, the extent of collateral vascular supply in the affected tissue,
- (2) Tissue's vulnerability to ischemia,
- (3) Caliber of the arterial BV occluded; in general, however, arterial emboli cause



Systemic Thromboembolism





<u>لوشتهن قاف خدام ويدنا نغوف سبب إوفاة -</u>

Postmortem Clots

- Postmortem Clots : At autopsy, postmortem (PM) clots may be mistaken for venous thrombi.
- PM clots are gelatinous with a dark red dependent portion where RBCs have settled by gravity, & a yellow chicken fat" supernatant; they are usually
 الم المرابع بيرك س دون المتعنى وسنما المرابع ال

 In contrast, red thrombi are firmer, almost always have a point of <u>attachment</u>, & on روز مراجعا روز مراجعا من مرجون مراجع مرجون مراجع مرجون مرجعا مرجون مرجعا مرجون مرجعا مرجون مرجعا مرجمع مرجعا م Post-mortem clot. Typically, a glistening, semi-translucent, homogeneous pale yellow (chicken-fat) clot which formed a cast of the pulmonary trunk & its branches, sometimes, they appear deep red (red current jelly clot). Post-mortem clots do not show lines of Zahn.



history : الأسئلة of car accidants & fraction & symptoms of embolism

Fat Embolism

Caused by:

- Soft tissue crush injury or long bone fractures, with release of microscopic fat globules into the circulation.
- Fat embolism occurs in some 90% of individuals with severe skeletal injuries, but less than 10% show any clinical findings.
 - Causes of fat embolism include
 - 1. Fracture of long bones
 - 2. severe burn
 - 3. severe fatty liver causing liver cirrhosis
 - 4. oily intravenous injections (mismanagement)->IM بنعطي عن طريق
 - 5. surgical operations (liposuction)

Fat embolism syndrome:

ئۆس سىريىغ

snortness of breath a. Pulmonary insufficiency (tachypnea, dyspnea)

b. Neurologic symptoms (irritability and restlessness to coma)

c. Anemia, thrombocytopenia. hemolysis ويعمل RBC& platelets ويتبط مع fat ال

d. Diffuse petechial rash

72 hours Typically, the symptoms appear 1 to 3 days after injury with sudden onset of symptoms

Fat Embolism

 \mathcal{D}

Pathogenesis: ^{السبب}

- Mechanical theory:
 - Mechanical obstruction by <u>microemboli of</u> <u>thrombus</u> <u>neutral fat</u> +platelet & RBC aggregates
 - Intravascular coagulation theory:
 - Chemical irritation (local injury to endothelium) from release of fatty acids + platelet activation & recruitment of granulocyyes –release of free radicals,protease &ecosanoids –>DIC

a characteristic petechial skin rash is related to rapid onset of thrombocytopenia, presumably caused by platelets adherence to the myriad (tens of thousands) fat globules & being removed from the circulation.

Adherence of platelets Thrombocytopenia (low platelet count) Bleeding tendency (petechial hemorrhage) Figure 50 - Fat embolism: Brain. Before his death, the patient had a fractured femur .At PM, coronal section of the frontal brain region shows multiple small hemorrhagic foci scattered throughout the white matter.



9.30 Fat embolism: brain

Amniotic Fluid Embolism السائل المحيط بالجنين

amniotic fluid و maternal circulation بالعادة ال circulation للأم والجنين ما برتبطوا مع بعض معن الخالط بين

Introduction of amniotic fluid and its contents to the maternal circulation via a tear in the placental membranes and rupture of uterine veins during childbirth

*الي بصير انه يدخل الamneotic على الcirculation تبع الأم وبالنسبة لجسم الأم هاد اشي غريب فراح يصير anphylactic shack

Rare (1 in 40,000 deliveries), but carries 80% mortality rate الأم * يوجد مطر ليرماح الأم

Brein//CNS // heart // lungs li it

Manifestations: <u>Respiratory failure</u> (sudden severe dyspnea, cyanosis, and hypotensive shock), seizures, and coma

Elements of baby's amneotic fluid

Histologic analysis: squamous cells shed from fetal skin,
 العبر lanugo hair, and mucin derived from the fetal respiratory or gastrointestinal tracts present in the maternal pulmonary
 الوير) microcirculation





Amniotic Fluid Embolism

Air embolism

- Source: air may enter the circulation
- (1) during obstetric procedures (tubal insufflation)
- (2) as a consequence of chest injury (stabbing)or neck stabbing by sharp tool causing puncture of internal jugular vein. Or artificial pneumothorax during operation.
- (3) miss management of intravenous infusion .
- (4) Scuba diving
- Generally, in excess of 30-50 mL of air is required to produce a clinical effect; the air bubbles act like physical obstruction (just as thromboembolism & causing distal ischemic injury), bubbles may coalesce to form frothy masses sufficiently large to occlude major vessel



Decompression sickness

 Decompression sickness Is a particular form of gas embolism, which occurs when individuals are exposed to sudden changes in atmospheric pressure. Scuba (under water breathing apparatus users) deep sea divers, underwater construction workers, & individuals in unpressurized aircraft in rapid ascent are at risk.





- When air is breathed at high pressure (e.g., during a deep sea dive) increased amounts of gas (particularly nitrogen) become dissolved in the blood & tissues.
- If the diver then ascends (depressurizes) too rapidly, the nitrogen expands in the tissues & bubbles out of solution in the blood to form gas emboli.
- Clinically, the rapid formation of gas bubbles within skeletal muscles & supporting tissues in & about joints is responsible for the painful arching of the backs, condition called (the bends)



Chokes, or pulmonary decompression sickness, is a rare but severe manifestation of decompression sickness (DCS) that can be rapidly fatal even with appropriate treatment. DCS and arterial gas embolism are

collectively referred to as decompression illness.



- Gas emboli may also induce focal ischemia in a number of tissues, including brain, heart, & in the lungs where it may leads to respiratory distress, called the chokes.
- Treatment of gas embolism consist of :
- 1. placing the individual in a compression chamber, where the barometric pressure may be raised, thus forcing the gas bubbles back into solution.
- 2. Subsequent, slow decompression, theoretically permits gradual resorption & exhalation of the gases so that obstructive bubbles do not reform.
- A more chronic form of decompression sickness is called Caisson disease, in which persistence of gas emboli in the bones leads to multiple foci of ischemic necrosis; the commonest sites are the heads of the femur, tibia, & humeri



INFARCTION

- Infarct: area of ischemic necrosis caused by occlusion of vascular supply in a particular tissue.
- Arterial thrombosis or arterial embolism underlies the vast majority of infarctions.
- Venous thrombosis can cause infarction, but it more often induces venous obstruction and congestion. blood & edema تجمع
- Infarcts caused by venous thrombosis thus usually occur only in organs with a single efferent vein (e.g., testis or ovary).

INFARCTION

 Infarcts are classified on the basis of their
 color (reflecting the amount of hemorrhage) and the presence or absence of microbial infection:

- Red (hemorrhagic)
- White (anemic)
- Septic.

Red infarcts

- (1) With venous occlusions (such as in ovarian التواء التواء torsion). hemorragic فيصير infraction مع انه عندي arterial supply
- (2) In loose tissues (such as lung).
- (3) In tissues with dual circulations such as lung and small intestine.= collection of blood
- (4) In tissues that were previously congested because of sluggish venous outflow.
- (5) When flow is **re-established** to a site of previous arterial occlusion. infracted مسكرة فبالتالي انا راح احط شبكيه بمنطقة *

مسكره فبالتالي أنا راح احط شبكية بمنطقة coronary artreles مسكره فبالتالي أنا راح احط شبكية بمنطقة aschemia لانه لاته الblood supply مسكرة فما حيوصل blood supply فحيكون عنا aschemia فانا راح احط شبكيه في منطقة infracted اصلًا فبالتالي راح يوصل blood supply لمنطقة ميتة فراح يبين red فيالحالة الطبيعية انا عندي أكثر من لو وقف سير الدم بواحد منهم الثاني بغذي المكان بس بس الي بصير انه التسكير لالsudden بكون nouden ما بلحق الثاني يغذي المنطقة فبصير infraction

Red infarcts





7.38 Infarction: lung

Figure 54 -Lung infarction. There is lower lobe,sub-pleura, pale pink, wedge-shaped infarct. The infarct is swollen, with raised pleural surface over it, & is surrounded by a dark-red congested border.

White infarcts ↓ shaped ↓ organs ↓ organs ↓ with end-arterial occlusions in solid organs with end-arterial circulations (e.g., heart, spleen, and kidney)

Where the solidity of the tissue limits the amount of hemorrhage that can seep into the area of ischemic necrosis from the adjoining capillary beds



Fig. 55 : A, Hemorrhagic wedge-shaped pulmonary (red infarct).

B, Sharply demarcated pale infarct in the spleen (white infarct).

Infarction

Infarcts tend to be wedge-shaped, with the occluded vessel at the apex and the organ periphery forming the base

 The main histological finding: ischemic coagulative necrosis, except the brain, in which liquefactive necrosis occurs. F 56 : Infarction: Brain. The patient had tentorial herniation obstructing the posterior cerebral arteries, which results in recent hemorrhagic infarction of the infero-medial aspects of both occipital lobes .



9.47 Infarction: brain

Shock

blood supply for all organs

 Definition: Systemic hypoperfusion and reduced oxygen delivery due to either reduced cardiac output, or ineffective circulatory blood volume.

• طيب اولًا بدك تعرف انه الblood pressure يعتمد على

شغلتين :الresistance والcardiac output

- Results of shock:
 - -hypotension.

•في حال قل الvasodilation الresistance راح يقل فبالتالي يقل الBP

• طيب عندك الcardiac output وهو العامل الثاني الى بيعتمد عليه الBP هاد بيعتمد على حاجتين برضه الheart rate والvolme في حال قل اي منها بقل الcardiac output فبقل الBP

- impaired tissue perfusion.
 * في حال عندي (heart attack) العضلات راح تموت فبقل
- cellular hypoxia.

الcontraction فبقل الvolume فبقل الcardiac output ويقل الBP

sympathatic --> epiniphrine --> vasoconstriction

 There are tow mechanisms to increase BP : kidney --> renin --> * Na & water retention



- Definition: Systemic hypoperfusion and reduced oxygen delivery due to either reduced cardiac output, or ineffective circulatory blood volume.
- is a life-threatening medical condition and is a medical emergency. If shock is suspected call 911 or get to an emergency department immediately.
- Results of shock:
 - Hypotension, impaired tissue perfusion.cellular hypoxia.
 - The main symptom of shock is low blood pressure. Other symptoms include rapid, shallow breathing; cold, clammy skin; rapid, weak pulse; dizziness, fainting, or weakness.

Major types of shock

- Cardiogenic shock: results from low cardiac output due to myocardial pump failure.
- Hypovolemic shock: results from low cardiac output due to loss of blood or plasma volume (e.g., due to hemorrhage or fluid loss from severe burns).
- Anaphylactic shock , caused by hypersensitivity or allergic reaction
- Neurogenic shock, Neurogenic shock is caused by <u>spinal cord injury</u>, usually as a result of a traumatic accident or injury.
- Septic shock
 Infections



Vasovagal syncope

- Vasovagal syncope is the most common cause of fainting.
- It happens when the blood vessels open too wide or the heartbeat slows, causing a temporary lack of blood flow to the brain.
- It's generally not a dangerous condition. To prevent fainting, stay out of hot places and don't stand for long periods





Septic shock



- High mortality rate
- Gram-positive bacteria constitute the most common cause of septic shock, followed by gram-negative organisms and fungi.
- Systemic arterial and venous dilation leads to tissue hypoperfusion.



Septic shock

- High mortality rate
- is a life-threatening condition that happens when your blood pressure drops to a dangerously low level after an infection. Any type of bacteria can cause the infection.
- Gram-positive bacteria constitute the most common cause of septic shock, followed by gram-negative organisms and fungi.
- Systemic arterial and venous dilation leads to tissue hypoperfusion.
- Septic shock is the last and most severe stage of sepsis. Sepsis occurs when your immune system has an extreme reaction to an infection. The inflammation throughout your body can cause dangerously low blood pressure. You need immediate treatment if you have septic shock. Treatment may include antibiotics, oxygen and medication.





Pathophysiology of Hypovolemic shock



Hypovolemic shock is a life-threatening condition that occurs when there is a severe decrease in blood volume in the body. It is typically caused by a significant loss of fluids, such as blood, due to various factors like trauma, internal bleeding, dehydration, or excessive fluid loss from conditions like severe diarrhea or vomiting.

The human body relies on an adequate volume of blood to deliver oxygen and nutrients to organs and tissues. When there is a significant reduction in blood volume, the heart is unable to pump enough blood to meet the body's needs, leading to a state of shock.

Some common signs and symptoms of hypovolemic shock include:

Rapid heart rate (tachycardia) and weak pulse Low blood pressure Rapid and shallow breathing Pale and cool skin Sweating and clammy skin Confusion or altered mental state Weakness and fatigue Thirst and dry mouth Decreased urine output

Hypovolemic shock is a medical emergency that requires immediate attention. Without prompt treatment, it can lead to organ failure and death.

Treatment typically involves restoring the blood volume and improving circulation.

This may include intravenous fluid resuscitation to replace lost fluids, blood transfusions in cases of severe blood loss, and identifying and treating the underlying cause of the shock.

It's important to note that hypovolemic shock is a serious condition, and if you suspect someone is experiencing it, you should seek immediate medical assistance or call emergency services.

Stages of Shock

Shock is a progressive disorder that leads to death if the underlying problems are not corrected

- Non-progressive phase: Compensatory mechanisms maintains perfusion of vital organs.
- Progressive phase: Tissue hypoperfusion with metabolic and circulatory worsening.
- Irreversible stage: Severe irreversible tissue and cellular injury that even if the hemodynamic defects are corrected, survival is not possible

- The clinical manifestations of shock depend on the precipitating insult.
- In hypovolemic and cardiogenic shock: hypotension, a weak rapid pulse, tachypnea, and cool, cyanotic skin.
- In septic shock: the skin may be warm and flushed owing to peripheral vasodilation.
- Prognosis varies with the origin of shock and its duration.
- substantially worse outcomes بسبه أكثر من

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microorgani
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Treatment

- Septic shock is treated with antibiotics and fluids.
- Anaphylactic shock is treated with diphenhydramine (Benadryl),
 2 epinephrine (an "Epi-pen"), and steroid medications (solumedrol).
- Cardiogenic shock is treated by identifying and treating the underlying cause.
- Hypovolemic shock is treated with fluids (saline) in minor cases, and blood transfusions in severe cases.
- Neurogenic shock is the most difficult to treat as spinal cord damage is often irreversible. Immobilization, anti-inflammatories such as steroids and surgery are the main treatments.
- Shock prevention includes learning ways to prevent heart disease, injuries, dehydration, and other causes of shock.



