SHOCK

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Objectives

To Undrestand :

- the definition of shock
- Pathophysiology of shock
- Types of shock
- How to diagnose shock
- Treatment /Resuscitation
- End point of resuscitation

- Definition : Shock is a systemic state of low tissue perfusion which is inadequate for normal cellular respiration. With insufficient delivery of oxygen and glucose, cells switch from aerobic to anaerobic metabolism.

Pathophysiology

- *Cellular*

As perfusion to the tissues is reduced, cells are deprived of oxygen and must switch from aerobic to anaerobic metabolism. The product of anaerobic respiration is not carbon dioxide but lactic acid causing lactic acidosis. there is failure of sodium/potassium pumps in the cell membrane and intracellular organelles. Intracellular lysosomes release autodigestive enzymes and cell lysis ensues. Intracellular contents, including potassium are released into the blood stream.

- *Microvascular*

ischaemia activate immune and coagulation systems. Hypoxia and acidosis activate complement and prime neutrophils, resulting in the generation of oxygen free radicals and cytokine release. These mechanisms lead to injury of the capillary endothelial cells. These, in turn, further activate the immune and coagulation systems. Damaged endothelium loses its integrity and becomes ‘leaky’. Spaces between endothelial cells allow fluid to leak out and tissue oedema ensues, exacerbating cellular hypoxia.

- *Systemic:*

Cardiovascular: compensatory baroreceptor response resulting in increased sympathetic activity and release of catecholamines into the circulation. This results in tachycardia and systemic vasoconstriction .

- Respiratory:

The metabolic acidosis and increased sympathetic response result in an increased respiratory rate and minute ventilation to increase the excretion of carbon dioxide (and so produce a compensatory respiratory alkalosis).

- Renal

Decreased perfusion pressure in the kidney leads to reduced filtration at the glomerulus and a decreased urine output. The renin–angiotensin–aldosterone axis is stimulated, resulting in further vasoconstriction and increased sodium and water reabsorption by the kidney.

- Endocrine

As well as activation of the adrenal and renin–angiotensin systems, vasopressin (antidiuretic hormone) is released from the hypothalamus in response to decreased preload and results in vasoconstriction and resorption of water in the renal collecting system. Cortisol is also released from the adrenal cortex contributing to the sodium and water resorption and sensitizing the cells to catecholamines.

Ischaemia–reperfusion syndrome

- hypoxia and local activation of inflammation. Further injury occurs once normal circulation is restored to these tissues. The acid and potassium load that has built up can lead to direct myocardial depression, vascular dilatation and further hypotension. The cellular and humoral elements activated by the hypoxia (complement, neutrophils, microvascular thrombi) are flushed back into the circulation where they cause further endothelial injury to organs such as the lungs and the kidneys. This leads to acute lung injury, acute renal injury, multiple organ failure and death.

Classification of shock:

- *Hypovolaemic shock:*

due to a reduced circulating volume. may be due to haemorrhagic or non-haemorrhagic causes. Non-haemorrhagic causes include poor fluid intake(dehydration), excessive fluid loss due to vomiting, diarrhoea,urinary loss (eg. diabetes), evaporation, or ‘third-spacing’ wherefluid is lost into the gastrointestinal tract and interstitial spaces, as for example in bowel obstruction or pancreatitis. Hypovolaemia is probably the most common form of shock.

- *Cardiogenic shock*

is due to primary failure of the heart to pump blood to the tissues. Causes include myocardial infarction, cardiac dysrhythmias, valvular heart disease, blunt myocardial injury and cardiomyopathy.

- *Obstructive shock*

there is a reduction in preload due to mechanical obstruction of cardiac filling. causes include cardiac tamponade, tension pneumothorax, massive pulmonary embolus or air embolus. In each case, there is reduced filling of the left and/or right sides of the heart leading to reduced preload and a fall in cardiac output.

Distributive shock (septic ,anaphylactic ,spinal)

- septic shock, anaphylaxis and spinal cord injury. Inadequate organperfusion is accompanied by vascular dilatation with hypotension, low systemic vascular resistance, inadequate afterload and a resulting abnormally high cardiac output. In anaphylaxis, vasodilatation is due to histamine release, while in high spinal cord injury there is failure of sympathetic outflow and adequate vascular tone (neurogenic shock). The cause in sepsis is less clear but is related to the release of bacterial products (endotoxin)

Endocrine shock

may present as a combination of hypovolaemic, cardiogenic or distributive shock. Causes :

hypo- and hyperthyroidism and adrenal insufficiency.. Cardiac output falls due to low inotropy and bradycardia. There may also be an associated cardiomyopathy. Thyrotoxicosis may cause a high-output cardiac failure. Adrenal insufficiency leads to shock due to hypovolaemia and a poor response to circulating and exogenous catecholamines. Adrenal insufficiency may be due to pre-existing Addison's disease or be a relative insufficiency due to a pathological disease state, such as systemic sepsis.

LECTURE 2

Severity of shock

- *Compensated shock*

reduced flow to non-essential organs to preserve preload and flow to the lungs and brain and kidney, by reducing perfusion to the skin, muscle and gastrointestinal tract.. Patients with occult hypoperfusion (metabolic acidosis despite normal urine output and cardiorespiratory vital signs) for more than 12 hours have a significantly higher mortality, infection rate and incidence of multiple organ failure .

- *Decompensation*
- Further loss of circulating volume overloads the body's compensatory mechanisms and there is progressive renal, respiratory and cardiovascular decompensation. In general, loss of around 15 per cent of the circulating blood volume is within normal compensatory mechanisms. Blood pressure is usually well maintained and only falls after 30–40 per cent of circulating volume has been lost.

- *Mild shock*

Initially there is tachycardia, tachypnoea, a mild reduction in urine output and the patient may exhibit mild anxiety. Blood pressure is maintained although there is a decrease in pulse pressure. The peripheries are cool and sweaty with prolonged capillary refill times

- *Moderate shock*

As shock progresses, renal compensatory mechanisms fail, renal perfusion falls and urine output dips below 0.5 mL/kg per hour. There is further tachycardia, and now the blood pressure starts to fall. Patients become drowsy and mildly confused.

- *Severe shock*

In severe shock, there is profound tachycardia and hypotension. Urine output falls to zero and patients are unconscious with laboured respiration.

- Capillary refill
- Most patients in hypovolaemic shock will have cool, pale peripheries, with prolonged capillary refill times. In distributive (septic) shock, the peripheries will be warm and capillary refill will be brisk, despite profound shock.

- Tachycardia

Tachycardia may not always accompany shock. Patients who are on beta-blockers or who have implanted pacemakers are unable to mount a tachycardia.

- Blood pressure

It is important to recognise that hypotension is one of the last signs of shock. Children and fit young adults are able to maintain blood pressure until the final stages of shock by dramatic increases in stroke volume and peripheral vasoconstriction. These patients can be in profound shock with a normal blood pressure. Elderly patients who are normally hypertensive may present with a ‘normal’ blood pressure for the general population but be hypovolaemic and hypotensive relative to their usual blood pressure. Beta-blockers or other medications may prevent a tachycardic response.

- **Consequences**
- *Unresuscitatable shock*

Patients who are in profound shock for a prolonged period of time become ‘unresuscitatable’. Cell death follows from cellular ischaemia and the ability of the body to compensate is lost. Death is the inevitable result.

Multiple organ failure

Multiple organ failure is defined as two or more failed organ systems . There is no specific treatment for multiple organ failure. Management is supporting of organ systems with ventilation, cardiovascular support and haemofiltration/dialysis until there is recovery of organ function.

- **Effects of organ failure**
- _ Lung: Acute respiratory distress syndrome
- _ Kidney: Acute liver insufficiency
- _ Clotting: Coagulopathy
- _ Cardiac: Cardiovascular failure

RESUSCITATION

- **Conduct of resuscitation**

Resuscitation should not be delayed in order to definitively diagnose. If there is initial doubt about the cause of shock, it is safer to assume the cause is hypovolaemia. In patients who are actively bleeding (major trauma, aortic aneurysm rupture, gastrointestinal haemorrhage), it is counterproductive to institute high-volume fluid therapy without controlling the site of haemorrhage. Increasing blood pressure merely increases bleeding from the site while fluid therapy cools the patient and dilutes available coagulation factors.

- **Fluid therapy**

In all cases of shock, regardless of classification, hypovolaemia and inadequate preload must be addressed before other therapy is instituted. Administration of inotropic or chronotropic agents .

Type of fluids

crystalloid solutions (normal saline, Hartmann's solution, Ringer's lactate) or colloids (albumin or commercially available products). there is little evidence to support the administration of colloids, which are more expensive and have worse side-effect profiles. Hypotonic solutions (dextrose etc.) are poor volume expanders and should not be used in the treatment of shock unless the deficit is free water loss (eg. diabetes insipidus) or patients are sodium overloaded (eg. cirrhosis).

Dynamic fluid response

250–500 mL of fluid is rapidly given (over 5–10 minutes). Patients can be divided into :

- **Responders** have an improvement in their cardiovascular status which is sustained. These patients are not actively losing fluid but require filling to a normal volume status.
- **Transient responders** have an improvement which then reverts to the previous state over the next 10–20 minutes. These patients have moderate ongoing fluid losses (either overt haemorrhage or further fluid shifts reducing intravascular volume).
- **Non-responders** are severely volume depleted and are likely to have major ongoing loss of intravascular volume, usually through persistent uncontrolled haemorrhage.

Vasopressor and inotropic support

not indicated as first-line therapy. administration of these agents in the absence of adequate preload rapidly leads to decreased coronary perfusion and depletion of myocardial oxygen reserves. Vasopressor agents (phenylephrine, noradrenaline) are indicated in distributive shock states (sepsis, neurogenic shock),vasopressin is alternative. In cardiogenic shock The dobutamine is the agent of choice.

Monitoring

- **Monitoring for patients in shock**
- Minimum

_ ECG
_ Pulse oximetry
_ Blood pressure
_ Urine output

- Additional modalities

_ Central venous pressure
_ Invasive blood pressure
_ Cardiac output
_ Base deficit and serum lactate

- *Systemic and organ perfusion: best monitor is* urine output. The level of consciousness is an important marker of cerebral perfusion.
- Base deficit and lactate: sensitive for both diagnosis of shock and monitoring the response to therapy.
- Mixed venous oxygen saturation:is a measure of the oxygen delivery and extraction bythe tissues. High mixed venous saturations (>70 per cent) are seen in sepsis and some other forms of distributive shock. In sepsis, there is disordered utilization of oxygen at the cellular level, and arteriovenous shunting of blood at the microvascular level

End points of resuscitation

- patients have been resuscitated until they have a normal pulse, blood pressure and urine output.

THANK YOU