SHOCK

Shock is a condition in which the metabolic needs of the body are not met because of an inadequate cardiac output. If tissue perfusion can be restored in an expeditious fashion, cellular injury may be limited, but if treatment is delayed shock may become irreversible with cellular injury and metabolic dysfunction becoming widespread.

AETIOLOGY OF SHOCK

(1) ***Inadequate circulating blood volume***

* Develops from intravascular loss of salt and water, loss of plasma or loss of blood.
- Results in depletion of various capacitance bed and decreased ventricular-end-diastolic volume stroke volume and cardiac output.

(2) ***Loss of Autonomic control of the vasculature***.

* Develops from spinal cord injuries, regional anaesthetics or administration of autonomic blocking agents.
- Results in expansion of the vasculature, which leads to inappropriate perfusion causing pooling of blood in vascular beds that do not need it at the expanse of those that do.

(3) Impaired cardiac function

* Develops from compression of the heart or intrinsic abnormality of the heart or obstruction of either the pulmonary or systemic vasculature.
- Results in decreased ventricular end-diastolic volume and cardiac output.

(4) Vascular Impairment.

- Develops when endogenous or exogenous vasoactive metabolites are released into the circulation.
- This causes a reduced arteriolar and venous vasomotor tone
- Included in this category are shock associated with the Systemic Inflammatory Response syndrome (SIRS) or sepsis, anaphylaxis, adrenocortical insufficiency, and traumatic injuries.

TYPES OF SHOCK

Inadequate circulating blood volume

Hypovolemic shock

* The losses are primarily external
* Can be due to haemorrhage (haemorrhagic shock), loss of fluid in to the gut, or as a result of vomiting/diarrhoea and dehydration (Non-haemorrhagic hypovolemic shock).

Vascular Impairment.

(i) Traumatic shock

- Initial loss of blood or plasma (e.g. burns) externally.
- Later, there is internal loss of fluid arising from wound produced inflammatory mediators and disrupted endothelium.

(iii) Septic shock

- Due to inflammatory mediators and disruption of endothelium, there is a loss of plasma into the tissue primarily. The early stages are characterized by fever and cutaneous vasodilatation.
- Later followed by the classical signs of shock.

Loss of autonomic control of vasculature.

- Neurogenic shocks may be due to spinal cord injury, severe head injury, or spinal anesthesia leading to a loss of vascular tone in the tissues supplied by the nerves.

Impaired Cardiac function.

This can be intrinsic or extrinsic.

(1) **Intrinsic** mechanisms of cardiac dysfunction leading to shock (***Cardiogenic shock)*** include myocardial infarction, cardiomyopathy, valvular heart disease, cardiac rhythm disturbances, and myocardial depression from drug toxicity or trauma.

(2) **Extrinsic** mechanisms of cardiogenic shock produce cardiac dysfunction by compressive or obstructive means. *Cardiac compressive shock* may be due to pericardiac tamponade or tension pneumothorax which may cause external compression of the heart, which impedes diastolic filling and decreases cardiac output. *Cardiac obstructive shocks* are due to pulmonary embolism or systemic arteriolar constriction causing an increased vascular resistance, thereby limiting systolic ejection.

CLINICAL FEATURES.

HYPOVOLEMIC SHOCK

The symptoms and signs are progressively due to:

(i) Sign of adrenergic discharge to the skin.

(ii) Oliguria

(iii) Cardiac or/and Neurologic dysfunction.

Mild Shock (<20% of blood volume)

Clinical features are those of increased adrenergic discharge to the skin.

- Cool & clammy extremities

- Collapsed subcutaneous veins
- Sweating (occasionally)
- Thirst.
- BP is normal in the supine position.
- Urinary output is normal but urine may be concentrated

Moderate Shock (20-40%) (due to effect of vasopressin and aldosterone).

- Oliguria
- Other signs of mild shock
- Supine B.P. is usually normal

Severe Shock (>40%) due to cardiac and neurologic dysfunction

- Low B.P. and IPR
- Change in the sensorium (Restlessness, stupor or unconsciousness.

Traumatic shock

- Initial signs and symptoms are due to external blood loss as in hypovolemic shock.
- But the major consequences are not shown until 24-48hrs by which the inflammation mediated vascular permeability would have developed. This aggravates pre-existing hypovolemia due to a generalized increase in systemic vascular permeability.

Septic shock

- Initial manifestation is due to attempt by the body to dissipate excessive heat produced by the hypermetabolic state viz a viz warm, pink and sweaty skin (early or red shock)
- Later hypoperfusion of vital organs activate cutaneous pressure mechanisms leading to the classical hypovolemic signs. There is a fall in skin temp which progresses to chills. The cutaneous vasoconstriction compromises the body's inability to dissipate heat and loads to another rapid rise in body temperature, which in turn leads to cutaneous vasodilatation.
- Terminally, there is widespread disruption of vascular endothelium leading to hypovolemic and reduction in cardiac output ("white or late septic shock).

Neurogenic shock

- History of spinal injury, regional anaesthetist or autonomic blocker administration.
- The skin of the denervated skin is warm and pink.
- BP is low.
- Heart Rate is rapid (or else slow if the adrenergic supply to the heart is block

- Heat loss can be considerable.

Cardiac compressive shock.

- Distended neck veins in injured patients with features consistent with hypovolemic shock are diagnostics of cardiac compressive shock.
- Clinical features of cardiac tamponade or tension pneumothorax.

Cardiac Obstructive shock

- Produces mechanical or functional obstruction that results in increased left ventricular or right ventricular afterloa causing failure..
- Coarctation of the aorta and malignant hypertension are examples of obstructive causes of increased left ventricular afterload, whereas pulmonary embolism and various other causes of pulmonary hypertension lead to increased right ventricular afterload and right ventricular failure.

Cardiogenic shock

- Feature of cardiac dysfunction co-existing with shock symptoms.

Treatment

General

- Airways maintenance
- Breathing
- Analgesia
- Cover the patient with blanket to prevent hypothermia.
- Supplementary oxygen therapy

Specific Treatment

Hypovolemic shock/Traumatic shock

- Infusion of crystalloids
- Blood infusion in haemorrhagic shock.

Septic shock

- Best prevented.
- Infusion of crystalloid
- Antibiotics.

Neurogenic shock

- Infusion of crystalloids
- Vasoconstrictor drugs e.g. Norepinephrine
- Elevation of the part of the body that is involved.

Cardiogenic shock

- Optimise the heart rate

- Vasodilators e.g. dopamine
- May require cardiac surgery.

Cardiac Obstructive shock

- Treat the underlying cause

Monitoring the patient

- Serial haematocrit
- Hourly urinary output monitoring
- Intra-arterial catheters to monitor arterial BP and arterial blood gases.
- Swanz-Ganz catheters to monitor the arterial wedge pressure.

Organ failure syndromes of shock

LUNGS

- Most of the late deaths after an initial successful resuscitation from shock are caused by respiratory failure.
- Usually due to respiratory distress syndrome.
- May become manifested within 24hrs of injury.
- Early signs are tachypnea and increased respiratory effort.
- Underlying cause is due to widespread inflammation of coagulopathy leading to microvascular damage.

KIDNEYS

- In shock, blood shunts to the medulla and bypasses the cortex resulting in cortical necrosis leading to renal failure if prolonged.

LIVER

- Shock induced liver failure is usually manifested by elevation of serum bilirubin and liver enzymes.

GIT

- Ischaemic damage of the mucosa occurs (called stress ulcer in the stomach). This may allow bacterial endotoxin to reach the circulation.

ADRENALS

- Acute adrenal insufficiency can result from haemorrhagic necrosis of the adrenals in leading to refractory shock.

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