Distributive shock

Distributive shock is characterized by a **decreased peripheral vascular resistance**, or **generalized vasodilatation** causing fluid to leak out of the vessels. The vasodilatation is not related to tissue hypoxia or an increased demand of nutrients, but it is caused by pathogenic shock-inducing stimulus e.g., bacterial toxin in septic shock or degranulation products of allergen-stimulated mast cells in anaphylactic shock.

Division

1. Septic-toxic shock
2. Anaphylactic shock
3. Neurogenic shock
4. Endocrine shock

Septic-toxic shock

Cause

- Bacterial infection, usually preceded by bacteremia (septic shock is sepsis (i.e. SIRS due to infection) with persistent arterial hypotension)

Pathogenesis

The **production of endotoxin** (lipopolysacharide G–) or **exotoxin** G+ and **pro-inflammatory cytokines** (IL-1, TNF-α) as a result of infection, stimulate the endothelium and macrophages. The gene for inducible NO-synthase is expressed and **NO** is produced, which causes **generalized vasodilation** and is followed by a decreased arterial pressure and a decreased afterload. Thus, the **ejection fraction and stroke volume of LV will increase**. Hypotension causes sympathoadrenal activation, which further increases myocardial contractility and heart rate which causes **cardiac output to increase** (in contrast to hypovolemic and cardiogenic shock), hence the name **hypercirculatory shock**.

In **septic shock** despite the increased organ perfusion **similar changes as in hypovolemic or cardiogenic shock occur**. The cause may be the bypassing of the own nutritional microcirculation by arterio-venous junctions, which also open during generalized vasodilation or an increase in pH (resulting from respiratory alkalosis from hyperventilation, which is caused by hyperpyrexia from flushing out the endogenous pyrogen IL-1), during which it is more difficult to release oxygen from hemoglobin.

In the later stages of shock, the contractility of the myocardium decreases (the cause is not exactly clear, it is referred to as myocardial depression factor) and the permeability of the capillary wall increases, causing fluid to leak from the vascular bed into the interstitium. Both of these facts further worsen the arterial hypotension.

Further complications of septic shock are the development of **DIC** due to the generalized inflammation, as well as thrombi formation due to a decreased blood flow in the dilated parts of the vascular bed, which are especially dangerous in the pulmonary circulation where they impair the oxygenation of venous blood.

Clinical picture

In the early phase, so-called **warm hypotension** can be seen (the extremities are reddened, warm and dry).

The late phase is characterized by a decreased myocardial contractility which causes further worsening of hypotension. Furthermore, there is an increase in the permeability of capillaries, which leads to the leakage of fluids from the vascular bed into the interstitium, which leads to a decrease in the circulating blood volume and further exacerbation of the hypotension.

Prognosis

- **Poor**, mortality 50%

Anaphylactic shock

Cause

- Parenteral antigen penetration

Pathogenesis

Antigen binds to IgE which is bound on the surface of mast cells and causes their degranulation. The released mediators (histamine, serotonin, prostaglandins, etc.) cause vasodilation and increase the permeability of capillaries, thereby decreasing cardiac output and causing arterial hypotension. **Complications** can be other

manifestations of allergy (Antigen action on other tissues), especially swelling of the airways and bronchospasm.

Prognosis

- Good

Main differences between septic and anaphylactic shock:

Type of shock	Progress	Prognosis	Cause
Septic	Slow	Bad	Infectious agent
Anaphylactic	Rapid	Good	Non-infectious agent

Neurogenic shock

Neurogenic shock occurs when the autonomic system is dysregulated due to spinal cord damage. The most common cause of spinal cord trauma is transverse spinal cord injury. Other less common causes are myelitis, toxins affecting CNS, spinal anesthesia or Guillain-Barré syndrome. In children, it can rarely occur with trisomy 21, skeletal dysplasia or tonsillopharyngitis. In the case of trauma, it is diagnosed *per exclusionem*, as the more frequent cause of shock is definitely hemorrhage.

Pathophysiologically, sympathetic activity is limited in case of damage above the vertebral level Th6 (more often in the thoracic spine area) or by a combination of primary damage (vertebral displacement/fracture) and secondary damage to sympathetic fibers (edema, devascularization, ...). Thus, the tone of the parasympathetic system does not have a natural opposition in its effect on the heart and blood vessels, and systemic circulation disorders occur.

As therapy, the basis is fluid support and possibly the use of vasopressors to maintain systemic pressure. This will also prevent secondary damage to the spinal cord. Another integral part is the immobilization of the cervical spine and subsequent consideration of neurosurgery. [1]

Links

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Related articles

- Shock
  - Cardiogenic shock
  - Obstructive shock
  - Hypovolemic shock
- Circulatory collapse

References

1. SAGAR, Dave – CHO, Julia J. *Neurogenic Shock* [online]. StatPearls Publishing, ©2021. [cit. 2021-04-11]. <https://www.ncbi.nlm.nih.gov/books/NBK459361/>.

Source

- PASTOR, Jan. *Langenbeck's medical web page* [online]. [cit. 30.5.2010]. <https://langenbeck.webs.com/>.

Distributive shock

Table comparing different types of shock