LECTURE 12: THE SHOCK

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- Shock is an imbalance between oxygen delivery (DO2) and oxygen demand can result from a reduction in oxygen delivery, maldistribution of blood flow, impaired oxygen utilization or an increase in tissue oxygen requirements
- Left unchecked, shock will result in a fall in oxygen consumption (VO2), anaerobic metabolism, tissue acidosis and cellular dysfunction leading to multiple organ dysfunction and ultimately death.

Classification of shock

1. Hypovolemic shock
   - This is probably the most common and most readily corrected cause of shock in surgical practice, and results from a reduction in intravascular volume secondary to the loss of blood (e.g., trauma, gastrointestinal hemorrhage), plasma (e.g., burns) or water and electrolytes (e.g., vomiting, diarrhea, diabetic ketoacidosis)
   - Causes of hypovolemic shock
     1. Gastrointestinal hemorrhage
        - Esophageal bleeding
        - Gastric and duodenal ulceration and bleeding
        - Cancer
        - Trauma
     2. Obstetric hemorrhage
     3. Pulmonary hemorrhage
        - Pulmonary embolus
        - Cancer
        - lung lesions, e.g., tuberculosis
        - Vasculitis
     4. Major blood loss during surgery.
2. Septic shock
   - Septic shock results from circulatory and cellular abnormalities occur as part of a dysregulation in host response to infection.
   - These changes impair tissue oxygen delivery.
   - Significant mortality rate (>40%).
   - The 1992 consensus definitions of sepsis (systemic inflammatory response syndrome [SIRS], sepsis, severe sepsis and septic shock) lack sensitivity and specificity.

- New definitions of (Sepsis-3) used a quick sepsis-related organ failure assessment (qSOFA) score to assess the presence of three symptoms:
    1. Altered mental status.
    2. Low blood pressure (<100 mmHg).
    3. Tachypnea (respiratory rate >22 breaths per minute).
- If a patient with infection has two or more of these criteria (qSOFA positive) they should be assumed to have sepsis.
- If qSOFA positive status should investigate for organ dysfunction and give therapy, including critical care referral، as appropriate.
- Sepsis usually arises from a localized infection, with
    1. Gram negative bacteria (38%).
    2. Gram positive bacteria (52%).
- The most common sites of infection leading to sepsis are
    1. The lungs (50–70%).
    2. Abdomen (20–25%).
    3. Urinary tract (7–10%).
    4. Skin.
- Cardiac output typically increases in septic shock to compensate for peripheral vasodilatation.
- Compounding disturbances in oxygen delivery, mitochondrial dysfunction may block the normal bioenergetic pathways within the cell, impairing oxygen utilization.
- Septic shock: severe sepsis where circulatory and cellular changes:
    1. Persistent hypotension requiring vasopressors to maintain mean arterial blood pressure > 65 mmHg.
    2. Serum lactate >2 mmol/L despite adequate fluid resuscitation.

3. Cardiogenic shock
    - This occurs when the heart is unable to maintain a cardiac output sufficient to meet the metabolic requirements of the body.
    - This ‘pump failure’ can be caused by myocardial infarction, arrhythmias, valve dysfunction, cardiac tamponade, massive pulmonary embolism and tension pneumothorax.

4. Anaphylactic shock

- This is a severe systemic hypersensitivity reaction following exposure to an agent (allergen) triggering the release of vasoactive mediators (histamine, kinins and prostaglandins) from basophils and mast cells.
- Anaphylaxis may be immunologically mediated (allergic anaphylaxis) or non-immunologically mediated (non-allergic anaphylaxis).
- The clinical features of allergic and non-allergic anaphylaxis may be identical, with shock a frequent manifestation of both.
- Anaphylactic shock results from vasodilatation, intravascular volume redistribution, capillary leak and a reduction in cardiac output.
- Common causes of anaphylaxis include
    1. Drugs (e.g., neuromuscular-blocking drugs, β-lactam antibiotics).
    2. Colloid solutions (e.g., gelatin containing solutions, dextrans).
    3. Radiological contrast media.
    4. Foodstuffs (peanuts, tree nuts, shellfish, dairy products).

5. Neurogenic shock
    - This is caused by a loss of sympathetic tone of vascular smooth muscle.
    - This typically occurs following injury to the thoracic or cervical spinal cord and results in profound vasodilatation, a fall in systemic vascular resistance (SVR) and hypotension.
    - A temporary drug-induced form can also occur in 'high' spinal anesthesia.

Clinical effects of shock

1. Nervous system
    a. Restlessness, confusion, coma.
    b. Encephalopathy and/or delirium.
2. Renal
    a. Renal hypoperfusion>> activation of the renin–angiotensin system.
    b. Oliguria (<0.5 mL/kg/h urine)>>> anuria
    c. Acute renal failure and metabolic acidosis
3. Respiratory
    a. Tachypnea and hypoxia

   b. Pulmonary edema (in cardiogenic shock)>>>hypoxia
   c. Acute respiratory distress syndrome>>>hypoxia
4. Cardiovascular
   a. ⬇Diastolic pressure >>> ⬇ coronary blood flow
   b. myocardial ischemia and infarction.
   c. Acidosis, electrolyte disturbances and arrhythmia.
5. Gastrointestinal
   a. Splanchnic hypoperfusion >> breakdown of gut–mucosal barrier.
   b. Stress ulceration.
   c. Translocation of bacteria.
   d. Acute ischemic hepatitis.

Management

1. General principles:
   a. The management of shock is based upon the following principles:
      i. Identification and treatment of the underlying cause
      ii. Resuscitation and the maintenance of adequate tissue oxygen delivery.
2. As clinical emergencies, treatment and diagnosis should occur simultaneously with immediate assessment and management following an Airway, Breathing, Circulation (ABC) approach.
3. Airway and breathing
   a. Hypoxemia must be prevented and rapidly corrected by maintaining a clear airway (e.g., head tilt, chin lift) and administering high-flow oxygen (e.g., 10–15 L/min).
   b. The adequacy of this therapy can be estimated continuously using pulse oximetry (SpO2), arterial blood gas analysis.
   c. Ventilator support: severe hypoxaemia, cardiovascular instability, depressed conscious level or exhaustion, intubation.
4. Circulation:
   a. Initial resuscitation should be targeted at arresting hemorrhage and providing fluid (crystalloid or colloid) to restore intravascular volume and optimize cardiac preload.

b. It is common practice to use blood, particularly if there is active bleeding.
c. As resuscitation continues, more invasive monitoring (central venous catheter; arterial line) allows the acid–base status, arterial and central venous pressure (CVP) to be used to further assess the response to fluid.
d. If blood pressure remains low then inotropes and/or vasopressors may be required.