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Chapter 1

Current Trends In Evaluation and Treatment of A Patient With Shock

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1. Introduction

Shock is defined as hypoperfusion of organs due to imbalance between demand and supply of oxygen. Shock is a medical emergency that needs early recognition. Main clinical features include hypotension, tachycardia, cool or warm extremities depending on the type of shock, tachypnea and pallor. Broadly, there are four types of shock i.e, hypovolaemic, distributive, cardiogenic and obstructive shock. Each type has different causes. For example, hypovolaemic shock may be caused by GI losses due to diarrhoea, vomiting or GI bleed. It may also be caused by haemorrhage. Distributive shock may be caused by sepsis, anaphylaxis, severe burns, syncope or pancreatitis. Cardiogenic shock may be caused by underlying cardiac conditions like arrhythmias, myocardial infarction, myocarditis and severe mitral or aortic valve insufficiency. Obstructive shock may be caused by pulmonary embolism, constrictive pericarditis and tension pneumothorax. Early recognition and determining the type of shock are key principles to initiate appropriate therapy. Initiation of the therapy should be simultaneous with looking into the cause of the shock.

2. Evaluation of The Patient

2.1. History

A concise and relevant history looking into the cause of shock should be obtained. If the patient is unconscious, the history should be obtained from accompanying family members or friends. In many cases type of shock is obvious from history for example, history of multiple episodes of vomiting indicates hypovolaemic shock. Similarly a history of trauma leading to haemorrhage will point towards hypovolaemic shock. A history of arrhythmias or coronary artery disease or underlying cardiac disease will point towards cardiogenic shock. In arrhythmias there will be complaints of syncope or presyncope. In case of coronary artery disease patient will give history of exertional chest pain. A history of fever and focal infection (e.g, burning during micturition, cough or abdominal discomfort) will point towards septic shock which is a type of distributive shock. History of dyspnoea and acute chest pain in an immobile patient will point towards obstructive shock due to pulmonary embolism.

2.2. Physical Examination

Physical examination will also help to find out the type of shock. Most commonly seen signs in shock are as follows.

1. **Hypotension:** A systolic blood pressure < 90 mmHg or mean arterial pressure < 65 mmHg suggests hypotension. Check that hypotension is not due to any prescribed medications.
2. **Tachycardia:** It is a compensatory mechanism in shock so, seen frequently. If there is no hypotension with tachycardia we can't deny shock because many young patients compensate for hypotension for extended time. In cardiogenic shock due to heart block there will be no tachycardia. There may be other cases of shock without tachycardia.
3. **Tachypnoea:** It is a common finding in shock due to increased oxygen demand because of underlying hypoxia.
4. **Confusion:** Decreased oxygen supply to brain may manifest as confusion. It may progress to encephalopathy.
5. **Urine Output:** We must assess urine output to rule out oliguria ($<$400ml/ Day or < 0.5 ml/kg/hr). If the patient is not micturating then insert a urinary catheter to monitor urine output.
6. **JVP:** The JVP may be elevated in cardiogenic shock with right sided heart failure. In contrast, JVP will be reduced in hypovolaemic shock due to low blood volume. Elevated JVP with pulsus paradoxus may indicate cardiac tamponade
7. **Cool or Warm extremities:** Warm extremities are found in only cardiogenic shock due to increased cardiac output. Cold, clammy extremities are found in rest all the types of shock.

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8. **Pulse pressure:** Wide pulse pressure is seen in cardiogenic shock and narrow pulse pressure is seen in hypovolaemic shock.

2.3. Relevant Investigations

1. **Serum Lactate levels:** Increased lactate levels are common in shock patients. Lactate is produced due to anaerobic glycolysis. Due to hypoxia of shock, TCA cycle can not oxidize pyruvate thereby converting pyruvate to lactate with the help of enzyme lactate dehydrogenase. In case of shock, because of hypoxia tissues produce increased amount of lactate.
2. **RFT:** BUN and serum creatinine provide risk assessment of kidney damage due to shock. Fractional excretion of sodium may provide an assessment of hypovolaemia.
3. **LFT:** Increased levels of alkaline phosphatase in blood may indicate obstructive jaundice due to biliary stones. This may indicate a source of infection to rule out septic shock.
4. **Cardiac Enzymes:** Increased levels of cardiac enzymes may point towards myocardial ischemia or myocarditis.
5. **Arterial Blood Gas (ABG) analysis:** ABG shows metabolic acidosis in most types of shock.
6. **Blood culture, Sputum culture and Urine culture:** These are done when septic shock is suspected.
7. **ECG:** ECG is done to identify any arrhythmias or MI as a cause of cardiogenic shock.
8. **Echocardiography:** It may rule out any valvular heart diseases, cardiac tamponade or ventricular dysfunction.
9. **Chest X-ray:** An interstitial infiltrate or alveolar infiltrate will indicate an infectious etiology like septic shock. Cardiomegaly, prominent pulmonary veins in upper lobes and peribronchial cuffing are suggestive of pulmonary oedema due to cardiogenic shock.
10. **CBC:** White blood cells are increased in infectious etiology.
11. **PT, INR:** Is important to know when we have to consider for thrombolysis.

3. Initial Management of Undifferentiated Shock

If you are unable to rule out etiology of shock from history or physical examination and lab investigations are awaited then you have to do some initial management. Prefer an ICU setting for shock treatment. I.V. access with two peripheral venous catheters will help in volume resuscitation with required fluids. For patients with persistent hypotension despite adequate fluid infusion central venous catheter (CVC) should be applied. CVC will help in central venous oxygen saturation monitoring and hemodynamic monitoring. We can also give drugs like inotropes through it. An arterial line placement will help in intravascular BP measurement. It will also help in determining accurate arterial oxygen saturation and acid base status of the patient. A urinary catheter should be placed to know the urine output of the patient. If required to maintain airway intubate and mechanically ventilate the patient.

4. The Need for Intravascular Volume Resuscitation

The volume status can be assessed by CVP line or by checking inferior vena cava diameter by Echocardiography. Recent studies have suggested that most patients with any of the four types of shock will always benefit from volume resuscitation. Benefit of volume replacement in distributive and hypovolaemic shock is clearly established. In cardiogenic shock patients there must be careful assessment of volume status before volume replacement. Even these patients will benefit from volume replacement. In septic shock a minimal volume replacement is required.

The volume replacement is most commonly done with crystalloid bolus (e.g, 500ml). In haemorrhagic shock with ongoing haemorrhage packed RBC must be given. Packed RBC should also be given if Haemoglobin < 7 gm/dl. The patient can be kept in Trendelenburg position temporarily for one minute and cardiac output and pulse pressure is assessed immediately. Echocardiography is also used increasingly nowadays to assess intravascular volume status. It must be done by trained personnel only.

5. Inotropic Drugs and Vasopressors

If hypotension persists after volume resuscitation then consider inotropic drugs or vasopressors as first line agents. For example, Norepinephrine is the first line drug in distributive shock because it acts on alpha 1 adrenergic receptors to cause vasoconstriction and on beta1 receptors to produce positive inotropic effect. Some studies support vasopressin as a second line agent in distributive shock as it can reverse vasodilation caused by distributive shock. Similarly, Dobutamine is a first line drug in cardiogenic shock. It acts via beta 1 and beta 2 receptors. Beta 1 effect produce positive inotropic effect and beta 2 causes decreased afterload. If BP does not increase adequately with dobutamine you should add norepinephrine to increase BP.

6. Oxygen Therapy

In all types of shock the patient can land in ARDS. Therefore supplemental oxygen should be kept ready. The aim is to maintain a saturation of 92 to 95%. Some patients may need to mechanical ventilation also.

Specific Therapies for Specific Type of Shock

1. **Hypovolaemic shock:** We have already discussed about fluid resuscitation in early management. Now, if there is massive haemorrhage consider blood transfusion. If there is GI bleeding consider endoscopic surgery to eliminate source of bleeding.
2. **Cardiogenic shock:** If the cause is acute coronary syndrome, rapid reperfusion (PCI) must be done. In case of right ventricular infarction intravascular volume replacement should be done to achieve a MAP of 10-15 mm Hg. If the cause is an arrhythmia the advanced cardiac life support should be given or artificial pacemaker if required.
3. **Distributive shock:** In case of sepsis broad spectrum antibiotics should be given after obtaining relevant cultures. In case of anaphylaxis remove the causative allergen, inject adrenaline and give vasopressors to combat vasodilation. In case of adrenal

insufficiency, steroids should be given in proper dose.

4. **Obstructive shock:** In case of pulmonary embolism consider thrombolysis or surgical clot removal. If the cause is tension pneumothorax consider mechanical decompression as the treatment.

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