Editorial

Circulatory Shock

"Shock occurs when there is circulatory failure that results in inadequate cellular oxygen, that is arterial blood flow is inadequate to meet tissue metabolic needs."

Tissue oxygenation is dependent upon, cardiac output, hemoglobin saturation and peripheral micro circulation.

Some or all of these factors may contribute to shock

In an attempt to maintain circulation to vital organs, the body responds to shock by release of catecholamines, rennin, antidiuretic hormone, cortisol and growth hormone. The variety of clinical manifestations that we encounter in shock, e.g. tachycardia, poor and delayed capillary refill, oliguria, restlessness, agitation and insulin resistance are but expression of these hormonal responses.

Clinically circulatory shock is recognized by signs of decreased tissue perfusion as evidenced by disturbance in function in certain organs and arterial hypotension i.e. BP less than 90mm systolic,(although in hypertensive shock patients it may be higher). Vasoconstriction causes cold and clammy skin, poor kidney perfusion leads to oliguria, (with fall in urine output to <0.5 ml per kilogram of body weight per hour). Confusion, disorientation and obtundation are caused by poor circulation to the brain.

Hypoxic cellular metabolism leads to increased serum lactic acid level which may aggravate the confusional state especially if the level is increased to more than 1.5 mmol per liter (normal blood lactate is approximately 1 mmol per liter).

Pathophysiology of shock

Hypovolemia may be caused by external fluid loss as in diarrhea and vomiting or with excessive sweating. Hemorrhage following trauma or from bleeding from GIT and from a massive hemoptysis causes hypovolemia and shock .Loss of plasma in burns is another example where severe fluid depletion can result in shock. Hyperosmolar states like diabetic ketoacidosis, or hyperosmolar non ketotic hyperglycemia also lead to severe fluid loss and shock.
Internal blood loss in a body space pleural or peritoneal or internal fluid redistribution as in pancreatitis, ascites or bowel obstruction are other pathologies which can end up in hypovolemic shock.

Cardiogenic factors which cause hypoperfusion are an increasingly important cause of shock in Pakistan.examples includes acute myocardial infarction, end-stage cardiomyopathy, advanced valvular heart disease, myocarditis, or cardiac arrhythmias.
Obstruction to circulation with resulting tissue hypoxia, as in pulmonary embolism, cardiac tamponade, aortic or mitral stenosis, atrial myxoma or tension pneumothorax, is another source of shock.

Distributive factors (e.g., severe sepsis or anaphylaxis from the release of inflammatory mediators**)** with decreased peripheral resistance and poor oxygen extraction play an important role in certain situations of shock

Neurogenic shock as induced by vasodilator drugs or Acute Adrenal Insufficiency can also result in shock.

Often times the patient may have a combination of these factors, e.g. patient with acute pancreatitis will be in acute distributive shock but may also have hypovolemic and cardiogenic shock from depression of myocardial function. This may also happen in anaphylaxis or sepsis.

In our country septic shock is probably the commonest form, although it may remain unrecognized till the late stage.cardiogenic shock will be a close second. Now that the number of road accidents is increasing especially involving motorcycles the hypovolemic shock will be encountered more often because of blood loss, internal or external. Because of awareness about ORS, fluid loss because of infantile diarrhea is less common, but still is a frequent cause of shock.

Septic shock, a form of distributive shock, is the most common form of shock among patients in the ICU especially in the extremes of age when immunity is low and ability to adjust to stress is poor. Cardiogenic shock is a common emergency as is hypovolemic shock. Obstructive shock is relatively rare. It may be kept in mind that condition of a patient with MI can easily worsen because of hypovolemia because of vomiting caused by pain and drugs and poor intake of fluids.

To make a diagnosis history could be most vital, although a history of a focus of origin of infection may have been forgotten or ignored by the patient. But in a clinical setting like in a diabetic or in patients with compromised immune system, infection as a possible cause of shock should be considered at the top.A typical history of myocardial infarction, arrhythmias or pulmonary embolism, valvular lesions like aortic stenosis will point to a possible cardiac origin. A history of trauma or crush injury or typical h/o pain of pancreatitis should straightaway lead to the diagnosis of hypovolemic shock.

Clinical signs like low blood pressure low thready pulse, irregular or pulsus paradoxus should provide a clue to the diagnosis.

Sometimes an investigation could be crucial in reaching a diagnosis. An ECG or an echocardiogram may point to the diagnosis of cardiac tamponade, pericarditis, or a valvular lesion (severe aortic mitral or pulmonary stenosis) or help in providing evidence for massive pulmonary embolism.

The type and cause of shock may be obvious from the medical history, physical examination, or lab investigations. For example, shock after traumatic injury is likely to be hypovolemic (due to blood loss).But cardiogenic shock or distributive shock may also occur, alone or in combination. A full clinical examination should include assessment of skin color and temperature, jugular venous pressure, and peripheral edema. The diagnosis can be refined, as mentioned above, with echocardiographic evaluation on arrival at emergency, which includes assessment for pericardial effusion, measurement of left and right ventricular size and function, and calculation of stroke volume.

Clinical assessment should include regular noting of blood pressure, Hypotension is defined as a systolic BP of less than 90mmHg or less, but consider the patients normal BP as the baseline. for example in a hypertensive patient a pressure of 100 may indicate shock A drop in BP of. 10-20mmHg and an increase in pulse rate of .15 with positional change is suggestive of low intravascular volume.

End organ hypoperfusion is judged by weak peripheral pulses, cool clammy or mottled extremities, oliguria, hepatic dysfunction or even bowel ischemia.Altered mental status should alert the keen observer to possibility of shock Patients may look normal or may be obtunded, restless agitated confused or comatose because of the poor brain perfusion.

Treatment

Basic life support promotes adequate circulation in addition to breathing through a clear airway (remember the mnemonic CAB which means Circulation, Airway and Breathing)
A useful mnemonic to describe the important components of resuscitation is the VIP rule. **Ventilate** (oxygen administration), **Infuse** (fluid resuscitation), and **Pump** (administration of vasoactive agents).

Certain basic principles must be kept in mind in treating shock

- Quick and effective hemodynamic support of patients in shock is crucial
- Resuscitation should be started immediately, even while investigation of the cause is going on.
- Once identified, the cause must be corrected rapidly.

  (e.g., control of bleeding, percutaneous coronary intervention for coronary syndromes, thrombolysis or embolectomy for massive pulmonary embolism, and administration of antibiotics and source control for septic shock).
- The latest advances in reduction in mortality of shock has been good observation of parameters of progress and improvement while the treatment is administered.
- Observe for rapid recovery signs, peripheral perfusion, pulse rate, blood pressure skin colour and breathing. Fingertip pulse oximetry should be the rule.
- If recovery is slow CVP line to monitor the Venous pressure to guide fluid therapy and for infusion of vasoactive agents.
- An arterial catheter should be inserted for monitoring of arterial blood pressure and blood sampling if the facilities are available. Mean arterial pressure should be maintained above 65mmHg.
- Generally a Central Venous Pressure or a Pulmonary Capillary Wedge Pressure (pcwp) of 5 mmHg suggests hypovolemia and a pressure of > 18 mmHg suggests volume overload cardiac failure, tamponade or pulmonary hypertension.

CARDIAC INDEX

The index is usually calculated using the following formula:
CARDIAC INDEX (**CI**) =CARDIAC OUTPUT / BODY SURFACE AREA WHICH IS EQUAL TO STROKE VOLUME X HEART RATE / BSA

A **CI**, <2 L / Min / M Sq. indicates a need for pharmacologic or mechanical pressor support. A high cardiac index (>4L / Min / M Sq) in a hypotensive patient is consistent with early septic shock.

Treatment is aimed to maintain a CVP of 8-12 mm Hg a mean arterial pressure of 65-90 mm Hg a cardiac index of 2-4 L / Min / M Sq. and CVP Oxygen saturation of more than 70%.

Volume replacement

In hypovolemic shock prompt replacement of fluids is required .Initially 2 liters of crystalloids may be given while assessment is being made of the degree of loss and the kind of fluid required. Helped by the CVP or PCWP and urine output findings.

Blood replacement as soon as possible is done where major cause is blood loss. When coagulation studies are abnormal platelets count is < 10,000/ mL or transfusion of > 6 units of blood is required, platelets and fresh frozen plasma is given.

Crystalloids like Normal saline or Ringer's lactate is given in amounts of 500 mL to 1000 mL when Hematocrit is > 30% and CVP is < 8mmHg. Glucose solution is generally used in hypoglycemia.

Plasma expanders / colloids such as albumen or dextran increase plasma osmotic pressure. They can cause coagulopathy or anaphylaxis, can increase pulmonary edema in SIRS. In a study reported in JAMA the investigators concluded, that among ICU patients with hypovolemia, the use of colloids vs crystalloids did not result in a significant difference in 28-day mortality. Although 90-day mortality was lower among patients receiving colloids, this finding should be considered exploratory and requires further study before reaching conclusions about efficacy. (*JAMA*. Published online October 09, 2013. doi:10.1001/jama.2013.280502).

Care should be exercised when larger amounts of fluids are being given, cold fluids can cause hypothermia.

Medications like pressor agents are given only after fluid loss has been replaced. In a Multicentre randomised matched trial reported in the new England journal of medicine which included 1679 patients, of whom 858 were assigned to dopamine and 821 to norepinephrine to restore and maintain mean blood pressure. The baseline characteristics of the groups were similar. Although there was no significant difference in the rate of death between patients with shock who were treated with dopamine as the first-line vasopressor agent and those who were treated with norepinephrine, the use of dopamine was associated with a greater number of adverse events. (N Engl J Med 2010;362:779-89.).The dosages used were, a dose of 20 µg per kilogram of body weight per minute for dopamine or a dose of 0.19 µg per kilogram per minute for norepinephrine, if blood pressure could not be maintained then open-label norepinephrine, epinephrine, or vasopressin could be added. The primary outcome was the rate of death at 28 days after randomization; secondary end points included the number of days without need for organ support and the occurrence of adverse events.

Dopamine effects are dependent on dosage. At low doses (2-3 micro grams /Kg /min) stimulation of dopaminergic and beta agonist receptors produces increased Glomerular Filtration Rate, heart rate and contractility. At higher doses (.5 micro grams /Kg /min) alpha adrenergic effects predominate causing peripheral vasoconstriction

Dobutamine (2-10 micro grams /Kg /min) a synthetic catecholamine has a greater ionotropic and after load reduction effects than dopamine. It is the first line drug for cardiogenic shock. Since tachyphylaxis occurs after 48 hrs .**Amrinone** (a phosphodiestrase inhibitor) (5-15micro grams /Kg /min) is used as replacement after 48 hours.
Distributive or Neurogenic Shock

These may need peripheral vasoconstrictors such as epinephrine (2-10 micro grams /min or nor epinephrine (0,5-30 micro grams / min)

ADH (Vasopressin) is being increasingly used in distributive shock. Its multiple effects include peripheral vasoconstriction, decreased heart rate, and hemostasis; increased serum cortisol and coronary cerebral and pulmonary vasodilatation.

Septic shock

Broad spectrum antibiotics are given in septic shock and treatment is started to cover the gram positive and gram negative organisms with broad spectrum antibiotics .Metronidazole is added if desirable to cover the anaerobes This regime is for the interim period till the results of blood culture and pus or tissue culture reports are received when the appropriate change in antibiotics is made.

Corticosteroids

Use of corticosteroids is controversial and probably not supported by randomised trials in shock. Its use in adrenal deficiency could be lifesaving

Corticosteroids are powerful anti-inflammatory agents. They may maintain vascular tone in states of shock. Endogenous cortisol is a stress hormone that acts in part to maintain vascular tone in states of shock. Some evidence suggests that exogenous hydrocortisone administration may increase mean arterial pressure and improve outcomes in patients with septic shock who have persistent hypotension despite adequate crystalloid resuscitation and vasopressin support. Dexamethasone may reduce sensorineural hearing loss in children and infants with H influenzae type B meningitis. Administer this agent to all children with suspected bacterial meningitis,

Recombinant Human Activated Protein C (Drotrecogin alfa)

Septic shock is a highly inflammatory and procoagulant state associated with significant mortality. APC is an endogenous protein that has natural anticoagulant and anti-inflammatory effects. In a single randomized controlled trial, recombinant human activated protein C (drotrecogin alfa) mortality in patients reduced with severe sepsis at high risk of death. In the Prowess-Shock trial, administration of recombinant human APC was not shown to improve mortality, and, therefore, the drug was withdrawn from the worldwide market on October 25, 2011.

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Critical Care Medicine

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(SIRS is systemic inflammatory response syndrome)

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