Management of septic shock in children

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Definition :

- Shock is a clinical syndrome of acute circulatory dysfunction , resulting in insufficient delivery of :
  1. Oxygen &
  2.Vital nutrients to tissues relative to their metabolic demand ,
  -which lead to cell death & organ dysfunction
  -Can lead to long term morbidity including neurological compromise.
- SEPTIC shock is defined as severe infection leading to cardiovascular dysfunction.

Physiology

- Oxygen delivered by heart to the cell is estimated by formula

$$DO_2 = CO \times (SaO_2 \times Hb \times 1.34) + (PaO_2 \times 0.003)$$

- So oxygen delivered to cell depend on three parameters:

  i) Cardiac output

  ii) Oxygen saturation in blood

  iii) Hemoglobin level

- Assuming a constant hemoglobin and PaO_2/SaO_2 ,value of cardiac output is the major determinant of oxygen delivery at most times
- Cardiac output is the product of stroke volume and heart rate. So it can be

  increased by increasing either heart rate or stroke volume

continued

In infant and children, the relative ability to increase cardiac output by increasing the heart rate is limited by

- preexisting elevated heart rate ,
- high level of connective tissues and
- low actin and myosin content in infant heart.

So cardiac output is mainly changed by change in stroke volume.

Stroke volume is dependent on

- Preload
- Cardiac contractility
- Afterload

Phases of shock and clinical features:

- Shock has three distinct but overlapping phases

1) Compensated phase :

- Redistribution of blood flow to vital organs (Heart , Brain & Adrenals) and away from non – vital organs (Skin , Gut , Kidney)
- Decreased peripheral perfusion- Cold extremities , acrocyanosis , pale skin with delayed CRT
- Blood pressure is preserved by increase in myocardial contractility & HR
- Stroke volume , CVP and UO decrease
- Weak peripheral pulses , Narrow pulse pressure & raised DBP)

- 2) Decompensated phase :
- Failure of compensatory mechanism
- Microvascular perfusion reduced- Lactic acid formation
- Myocardial contractility & BP decrease -decreased perfusion to vital organ
- Lack of perfusion to Brain- Change in Consciousness & lethargy
- 3) Irreversible phase :
- Tissue hypoxia with multi organ failure leading to death

Clinical parameter	`compensated	uncompensated	Irreversible
Mental state	Agitated or confused	Drowsy	**Unresponsive**
Heart rate	Tachycardia	Marked tachycardia	May develop bradycardia
Respiration	Normal or mild tachypnea	Tachypnea	Apnea
Skin and CRT	Increased CRT	Cold periphery with increased CRT	Increased CRT , cold cyanotic , **mottled skin**
Urine output	Adequate	OLIGURIA	Anuria
Blood pressure	Normal	**Hypotension**	**Unrecordable**

Types of shock

- HYPOVOLEMIC SHOCK
- CARDIOGENIC SHOCK
- OBSTRUCTIVE SHOCK
- DISTRIBUTIVE SHOCK
- SEPTIC SHOCK

Types of Shock

HYPOVOLEMIC	CARDIOGENIC	DISTRIBUTIVE	SEPTIC	OBSTRUCTIVE
Decreased preload secondary to internal or external losses	Cardiac pump failure secondary to poor myocardial function	Abnormalities of vasomotor tone from loss of venous and arterial capacitance	Encompasses multiple forms of shock Hypovolemic: third spacing of fluids into the extracellular, interstitial space Distributive: early shock with decreased afterload Cardiogenic: depression of myocardial function by endotoxins	Decreased cardiac output secondary to direct impediment to right- or left-sided heart outflow or restriction of all cardiac chambers
POTENTIAL ETIOLOGIES				
Blood loss: hemorrhage Plasma loss: burns, nephrotic syndrome Water/electrolyte loss: vomiting, diarrhea	Congenital heart disease Cardiomyopathies: infectious or acquired, dilated or restrictive Ischemia Arrhythmias	Anaphylaxis Neurologic: loss of sympathetic vascular tone secondary to spinal cord or brainstem injury Drugs	Bacterial Viral Fungal (immunocompromised patients are at increased risk)	Tension pneumothorax Pericardial tamponade Pulmonary embolism Anterior mediastinal masses Critical coarctation of aorta

What is sepsis?

- Microbiologically , Septicemia refers to the circulation and multiplication of bacteria in the blood with formation of toxic products and production of high fever.

- Clinically, Sepsis is defined as SIRS resulting from a suspected or proven infectious etiology.

What is SIRS?

- SIRS or Systemic Inflammatory Response Syndrome is defined as an inflammatory cascade that is initiated by the host response to an infectious or non-infectious trigger.

Criteria for SIRS

- Two of 4 criteria, 1 of which must be abnormal temperature or abnormal leukocyte count:
- Core temperature > 38.5oC (101.3oF) or <36oC (96.8oF)
- Tachycardia: Mean heart rate > 2SD above normal for age in absence of ext. stimuli, chronic drugs or painful stimuli, or Unexplained persistent elevation over 0.5- 4hr , or In children <1 year old, persistent bradycardia over 0.5 hr
- Respiratory rate >2 SD above normal for age or acute need for mechanical ventilation not related to NM disease or GA
- Leukocyte count elevated or depressed for age (not secondary to chemotherapy) or >10% immature neutrophils

Sepsis is defined as SIRS + Suspected or proven infection

Severe sepsis?

- Severe Sepsis is defined as sepsis plus any one of the following:

1.Cardiovascular organ dysfunction, defined as : Despite >40 ml/kg of isotonic IV fluid in 1 hour there is Hypotension or blood pressure < 5th percentile for age or SBP <2 SD below normal for age

or, Need for vasoactive drug to maintain blood pressure.

Or 2 of the following :

- Unexplained metabolic acidosis : base deficit > 5 meq/L
- Increased arterial lactate : > 2 times upper limit of normal
- Oliguria : urine output < 0.5 ml/kg/hr
- Prolonged capillary refill : >5 sec
- Core to peripheral temperature gap > 3oC (5.4oF)

Continued:

2. ARDS is defined by presence of PaO2/ FiO2 ratio < 300 mm Hg,

bilateral infiltrates on chest radiograph, and no evidence of left heart failure.

OR

Sepsis plus >=2 organ dysfunctions (respiratory, renal, neurologic, hematologic, or hepatic).

Septic shock

- Sepsis plus cardiovascular dysfunction as defined in above slide

Criteria for Organ Dysfunction

ORGAN SYSTEM	CRITERIA FOR DYSFUNCTION
Cardiovascular	Despite administration of isotonic intravenous fluid bolus ≥60 mL/kg in 1 hr: decrease in BP (hypotension) systolic BP <90 mm Hg, mean arterial pressure <70 mm Hg, <5th percentile for age, or systolic BP <2 SD below normal for age *or* Need for vasoactive drug to maintain BP in normal range (dopamine >5 µg/kg/min or dobutamine, epinephrine, or norepinephrine at any dose) *or* Two of the following: Unexplained metabolic acidosis: base deficit >5.0 mEq/L Increased arterial lactate: >1 mmol/L or >2× upper limit of normal Oliguria: urine output <0.5 mL/kg/hr Prolonged capillary refill: >5 sec Core-to-peripheral temperature gap: >3°C (5.4°F)
Respiratory	PaO_2/FIO_2 ratio <300 in absence of cyanotic heart disease or preexisting lung disease *or* $PaCO_2$ >65 torr or 20 mm Hg over baseline $PaCO_2$ *or* Need for >50% FIO_2 to maintain saturation ≥92% *or* Need for nonelective invasive or noninvasive mechanical ventilation

Neurologic	GCS score ≤11 *or* Acute change in mental status with decrease in GCS score ≥3 points from abnormal baseline
Hematologic	Platelet count <100,000/mm^3 or decline of 50% in platelet count from highest value recorded over last 3 days (for patients with chronic hematologic or oncologic disorders) *or* INR >1.5 *or* Activated prothrombin time >60 sec
Renal	Serum creatinine >0.5 mg/dL, ≥2× upper limit of normal for age, or 2-fold increase in baseline
Hepatic	Total bilirubin ≥4 mg/dL (not applicable for newborn) Alanine transaminase level 2× upper limit of normal for age

BP, Blood pressure; FIO_2, fraction of inspired oxygen; GCS, Glasgow Coma Scale; INR, international normalized ratio; $PaCO_2$, arterial partial pressure of carbon dioxide; PaO_2, partial pressure arterial oxygen; SD, standard deviations.

Pathophysiology of shock

EXTRACORPOREAL FLUID LOSS –Hypovolemic shock may be a result of direct blood loss through hemorrhage or abnormal loss of body fluid .

LOWERING PLASMA ONCOTIC PRESSURE-Hypovolemic shock may also result from hypoproteinemia.

ABNORMAL VASODILATION-Distributive shock occurs when there is loss of vascular tone – Venous , arterial or both .

INCREASED VASCULAR PERMEABILITY-Sepsis may change the capillary permeability in the absence of any change in capillary hydrostatic pressure.

CARDIAC DYSFUNCTION- Peripheral hypoperfusion may result from any condition that affects the heart’s ability to pump

Diagnosis and monitoring of shock

1.Shock is a clinical diagnosis based on a thorough history and physical examination.

2.Manifestation of shock consist of abnormalities in hemodynamic parameters and impaired end organ perfusion like

- tachycardia,
-cool extremities ,
-prolonged capillary refil time,
- poor peripheral pulses.

if present, strongly supports the diagnosis of shock which is a late feature.

4.Impaired capillary refill is a sensitive indicator of poor peripheral tissue perfusion

Continued

5.Blood lactate level [>2mmol/L] has a pivotal role in diagnosis of shock as it is a marker of decreased tissue perfusion and anaerobic metabolism.

6.Mixed venous oxygen saturation[SvO2] OR its surrogate i.e. superior vena caval oxygen saturation[Scvo2] is also an important marker of increased oxygen extraction and hence can be utilized for diagnosis and monitoring of shock.

Initial investigation

- CBC , CRP, ESR
- Blood sugar
- RFT,LFT and Serum electrolytes
- ABG , Blood lactate
- PT,aPTT,INR
- Blood culture & sensitivity.
- Chest X ray
- Echocardiography.

Treatment : Shock should be detected and managed before onset of hypotension

- **Supportive management**: correction of negative inotropic factor like hypoxia , acidosis .
- Early support of airway and breathing by non invasive or invasive ventilation with least work of breathing.
- Correction of metabolic derangement like hypoglycemia , hypokalemia , hypocalcemia.
- Administration of first line antibiotics in first hour of suspected septic shock.

Algorithm for management of pediatric septic shock

0 min - Recognize decreased mental status and perfusion .

- begin high flow oxygen and
- establish IO/IV access according to PALS.

5 min - If no hepatomegaly or rhonchi / crepts then push 20 ml/kg isotonic saline boluses and reassess after each bolus , upto 60ml/kg until improved perfusion . Stop if rhonchi , crepts or hepatomegaly.

- Correct hypoglycemia and hypocalcemia.
- Begin broad spectrum antibiotics.

Fluid refractory shock: 15min

- Begin peripheral IV/IO inotrope infusion , preferably epinephrine 0.05 -0.3 µg/kg/min.
- Use atropine /ketamine iv/io /im if needed for central vein or airway access.
- Titrate epinephrine 0.05-0.3µg/kg/min for cold shock .
- Titrate central dopamine 5-9 µg/kg/min if epinephrine not available .
- Titrate norepinephrine from 0.05µg/kg/min and upward to reverse warm shock.
- Titrate central dopamine ≥10 µg/kg/min if norepinephrine not available

Catecholamine resistant shock : 60 min

- If at risk of absolute adrenal insufficiency consider hydrocortisone .
- Use doppler USG ,
  - -PICCO (pulse index continuous cardiac output),
  - - FATD (femoral artery thermodilution) or
  - - PAC (pulmonary artery catheterization)

    to direct fluid , inotrope , vasopressor & vasodilators uses
- Goal is normal MAP & CVP , ScvO2>70 % and CI 3.3-6l/min/m2

Normal BP, cold shock

ScvO2 <70%/Hb >10g/dl on epinephrine ?

Begin Milrinone infusion. Add nitroso-vasodilator if CI<3.3 l/min/m2 with high SVRI and /or poor skin perfusion. Consider Levosimendan if unsuccessful.

low BP , cold shock

ScvO2 < 70 % /Hb > 10g/dl on epinephrine ?

Add norepinephrine to Epinephrine to attain normal diastolic BP. If CI < 3.3 l/min/m2 add Dobutamine, Enoximone , Levosimendan or Milrinone.

low Bp , warm shock

ScvO2 < 70% on norepinephrine?

If euvolemic , add Vasopressin, Terlipressin Or Angiotensin. But if CI decreases below 3.3l/min/m2 add Epinephrine, Dobutamine, Enoximone, Levosimendan.

Fluid refractory catecholamine resistant shock : ECMO

Refractory shock:

ALWAYS exclude-

-pericardial effusion

-Tension Pneumothorax

Therapeutic endpoints of treatment of septic shock

- Normalisation of HR, RR and mental status.
- CRT<2sec
- Warm extremities.
- Normal range of systolic BP & MAP
- Urine output >1ml/kg/hr
- Normal blood lactate & ScvO2>70%.

Cardiovascular Drug Treatment of Shock

DRUG	EFFECT(S)	DOSING RANGE	COMMENT(S)
Dopamine	↑ Cardiac contractility	3-20 µg/kg/min	↑ Risk of arrhythmias at high doses
	Significant peripheral vasoconstriction at >10 µg/kg/min		
Epinephrine	↑ Heart rate and ↑ cardiac contractility	0.05-3.0 µg/kg/min	May ↓ renal perfusion at high doses
	Potent vasoconstrictor		↑ Myocardial O_2 consumption
			Risk of arrhythmia at high doses
Dobutamine	↑ Cardiac contractility	1-10 µg/kg/min	—
	Peripheral vasodilator		
Norepinephrine	Potent vasoconstriction	0.05-1.5 µg/kg/min	↑ Blood pressure secondary to ↑ systemic vascular resistance
	No significant effect on cardiac contractility		↑ Left ventricular afterload
Phenylephrine	Potent vasoconstriction	0.5-2.0 µg/kg/min	Can cause sudden hypertension
			↑ O_2 consumption

Early initiation of antibiotics & source control

- Broad spectrum antibiotics adm. within 1 hr is important part of septic shock management in emergency.
- Blood culture needs to be sent before starting antibiotics.

Source control

- All sepsis child should be evaluated for the presence of focus of infection & it's T/T like

  -drainage of abscess,

  -debridement of wound &

  - Removal of intravascular devices etc.

Intervention to enhance host response

1) STEROIDS

-steroids should be used in suspected adrenal insufficiency

-surviving sepsis campaign 2020 guidelines recommends for the use of steroid in children with sepsis and sepsis induced organ dysfunction.

2) IVIG

- A randomized controlled study of polyclonal immunoglobulin in pediatric patients with sepsis showed reduction in mortality and hospital stay, however data are inadequate to support its regular use.

Supportive care

1) Mechanical ventilation-

-Early use of mechanical ventilation aided by sedation and paralysis.

-MV with PEEP improves oxygenation and decreases left ventricular afterload. NIV/IV

-It is recommended to intubate in fluid refractory shock.

-Rapid sequence intubation technique is preferred.

- **Blood products.**

-PRBC transfusion in a child with sepsis with Hb lesser or equal to 7gm/dl is preferred.

-Coagulation profile (PT, aPTT & INR) in a severe sepsis child should be done regardless of bleeding.

-FFP is only indicated in a patient with abnormal coagulation.

-Platelet transfusion should be done if <5000 without risk of bleeding and

PLT <30000 with risk of bleeding.

- **Glycemic control**

-Strict glycemic control

-Avoid blood glucose level >180mg/dl

-Low dose Insulin may be used to control.

Nutrition

- Adequate calorie maintenance
- Electrolytes imbalance management
- Early initiation of enteral feeding

Recent advances in septic shock management.

1) Plasmapheresis.
2) ECMO

PROGNOSIS

- **POOR PROGNOSTIC FACTORS**

-Younger age

-Child with immunodeficiencies.

-Site of infection

-Type of microrganisms

-MODS

-Multiple Vasoactive agents requirement.

Conclusions:

- The 1hr is a golden hr for the diagnosis and management of sepsis with shock.
- Early recognition & management of shock is of vital importance to limit the mortality and outcomes.
- Early initiation broad spectrum antibiotics.
- Goal directed management.
- Elevated blood lactate levels reflects poor tissue oxygen delivery noted in all form of shock.
- Early initiation of enteral feeding .


THANK YOU
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THE WALK TO CURE DIABETES
JDRF 2005
We're on Track For a Cure