CLINICAL PROFILE OF SHOCK IN CHILDREN IN A TERTIARY CARE HOSPITAL, MADURAI

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CERTIFICATE

This is to certify that the dissertation entitled **"CLINICAL PROFILE OF SHOCK IN CHILDREN IN A TERTIARY CARE HOSPITAL"** submitted by **Dr.V.K.VIJAYAMOHAN** to the faculty of Paediatrics, The Tamil Nadu Dr.M.G.R Medical University, Chennai in partial fulfillment of the requirement for the award of M.D. Degree Branch VII (Paediatrics) is a bonafide research work carried out by him under our direct supervision and guidance.

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TABLE OF CONTENTS

PROFORMA

Case No.:	IP. No.:	Outcome:
Name:	Religion:	Informant:
Age:	Occupation:	Father:
DOA:		
Sex:		Mother:
DOD:		
Address:		

CHIEF COMPLAINTS WITH DURATION

- Fever
- Loose stools
- Vomiting
- Pain abdomen
- Blood loss
- Skin bleeds (petechiae, purpura)
- Cough
- Chest pain
- Headache
- Convulsions
- Sensorium
- Convulsions
- Ear discharge

• Burning micturation

• Pyoderma

• Pain in joint

• Palpitation

• Cyanosis

• Cold extremities

• H/O suck rest suck cycle

• H/o drug intake

• H/o exposure to allergen

• H/o scorpion sting

• H/o snakebite

• H/o polyuria, polydypsia, weight loss

• H/o abrupt stopping of steroids

• H/o head injury

• H/o loss of weight, loss of appetite

PAST HISTORY

FAMILY HISTORY

BIRTH HISTORY: Antenatal history

Natal history

Post-natal history

Developmental History: Normal / Delayed

General physical examination

Anthropometry: Present Expected Comment

Weight (kg):

Length/height:

HC:

CC:

MAC:

Normal/grade: 1/2/3/4 IAP classification

Vitals:

HR (bpm) 0 hrs 12 hrs 24 hrs 48 hrs

Pulse

RR/min

BP (mm of Hg)

Temp (^{o}f)

CFT:

Urine output:

GCS:

Head to toe examination:

Head

Eye: Ear:

Mouth:

Neck: Hydration status:

Upper limb:

Lower limb:

Systemic examination

Cardiovascular system:

Inspection: Apical impulse

Precordial bulge

Other pulsation:

Palpation: Apex beat

Thrill

Parasternal heave-

Palpable p2

Percussion

Auscultation Mitral area:

Tricuspid area:

Pulmonary area:

Aortic area:

Per abdomen

Inspection:

Palpation:

Percussion:

Auscultation:

Respiratory system: Inspection: Trachea:

B/L chest movements:

Dilated veins:

Palpation:

Percussion:

Auscultation: Bilateral air entry

Breath sounds:

Added sounds:

Central nervous system

Higher mental function: GCS:

Cranial nerves:

Motor system: Bulk:

Tone:

Power:

Reflexes: Superficial

Deep

Gait:

Involuntary movements:

Sensory system

s/o meningeal irritation:

s/o cerebellar dysfunction:

Diagnosis: Functional category: Etiology:

Investigations: Complete blood count

Electrolytes: Na^+, K^+, Cl^-

Calcium, Phosphorus

RBS:

ABG:

Liver function test:

Renal function test:

Blood culture:

Urine Culture:

CSF analysis:

CXR: X-ray abdomen:

Ultrasound abdomen:

ECG: ECHO:

Treatment given:

IV fluids:

Antibiotics:

Vasopressors: Dopamine:

Dobutamine:

Epinephrine:

Norepinephrine:

O_2: l/min

Ventilator:

Specific Treatment:

LIST OF ABBREVIATIONS

ALT	→	Alanine Amino Transferase
AST	→	Aspartate Amino Transferase
ARDS	→	Acute Respiratory Distress Syndrome
ABG	→	Arterial Blood Gas
BP	→	Blood Pressure
CNS	→	Central Nervous System
CI	→	Cardiac Index
CRT	→	Capillary Refilling Time
CPTG	→	Core and Peripheral Temperature Gradient
CRP	→	C-reactive protein
ED	→	Emergency Department
DBP	→	Diastolic Blood Pressure
FIO_2	→	Fraction of Inspired Oxygen
GCS	→	Glasgow Coma Scale
IL-1	→	Interleukin-1
IL-2	→	Interleukin-2
IL-6	→	Interleukin-6
IL-8	→	Interleukin-8
IL-10	→	Interleukin -10
MAP	→	Mean Arterial pressure
NO	→	Nitric Oxide
PaO_2	→	Partial Pressure of Arterial Oxygen

PCO_2	→	Partial Pressure of Carbon Dioxide
PICU	→	Pediatric Intensive Care Unit
$PtcO_2$	→	Transcutaneous Partial Pressure of Oxygen
$PtcCO_2$	→	Transcutaneous Partial Pressure of Carbon Dioxide
PCT	→	Procalcitonin
SBP	→	Systolic Blood Pressure
SIRS	→	Systemic Inflammatory Response Syndrome
SpO_2	→	Saturation of Oxygen
SVR	→	Systemic Vascular Resistance
SVRI	→	Systemic Vascular Resistance Index
$SVCO_2$	→	Superior Venacaval Oxygen Saturation (mixed venous oxygen saturation)
TNF ALFA	→	Tumor Necrosis Factor - Alfa
WHO	→	World Health Organization

INTRODUCTION

Shock or circulatory failure is an acute syndrome characterized by inadequate circulatory perfusion of tissues to meet the metabolic demands of vital organs[1].

Shock is one of the commonest pediatric emergencies.[2] The shock states in children are classified according to the etiological factors namely hypovolemic shock due to volume depletion, cardiogenic shock due to cardiac dysfunction, distributive shock due to abnormal vasodilatation and septic shock due to increased vascular permeability.

Early recognition with efficient, anticipatory, and aggressive management of children in shock is mandatory and will often be rewarding, as early restoration of tissue perfusion to normalcy will determine the immediate outcome. The final outcome will depend upon the nature of etiology[2] and the availability of intervention measures.

These children with shock are often referred to tertiary care facility for admission and management. The time lapse between the onset of this state and the ·time of admission and initiation of resuscitative measures is a great factor in determining the outcome.[3] These children are looked after in a pediatric intensive care setup where constant observation and vigil

with appropriate monitoring of various clinical parameters and laboratory parameters will determine and modify the therapeutic intervention which in turn will determine the outcome.

In any pediatric setup, pediatric shock states are often secondary to hypovolemic states, envenomation secondary to scorpion sting, gram-negative septicemia, cardiac dysfunction, and anaphylactic reaction. This study is a prospective observational study of shock states in children beyond neonatal period (> 30 days to 12 years). As mentioned earlier, this is one of the commonest emergencies in pediatrics wherein the mortality rate is found to be quite significant. The mortality rate is extremely high in septic shock even in developed countries[2], where as the outcome in shock states secondary to envenomation is extremely gratifying. Hence this study is undertaken so as to find out the occurrence of this problem among pediatric admissions, the various causes contributing to them and to assess the outcome in relation to the various clinical and monitoring parameters.

AIM OF THE STUDY

1. To find out the incidence of shock states in pediatric age group in Govt. Rajaji Hospital, Madurai.
2. To categorize the shock states based on etiology.
3. To find out association of various clinical and monitoring paramerters of shock with outcome.

REVIEW OF LITERATURE

Shock is an acute, complex state of circulatory dysfunction that results in failure to deliver sufficient amounts of oxygen and other nutrients to meet tissue metabolic demands and, if prolonged, leads to multiple organ failure and death.[4] Shock states, therefore, may be viewed as a state of acute cellular oxygen deficiency. Shock is not a problem of blood pressure or blood volume, but, whatever the causative factors, it is always a problem of inadequate cellular sustenance.[5-7] Shock can be caused by any serious disease or injury; it is the final common pathway to death.

Delivery of oxygen is a direct function of the cardiac output and the arterial oxygen content. Inadequate oxygen delivery can result from either limitation or maldistribution of blood flow[8]. Occasionally increased oxygen requirements (fever, sepsis, or trauma) may result in cellular oxygen deficiency with normal blood flow and oxygen delivery. Reduced oxygen content (anemia, poor arterial oxygen saturation) requires higher cardiac output to maintain oxygen delivery.

When oxygen delivery fails to meet cellular oxygen demands, various compensatory mechanisms are activated. Shock, therefore, is a dynamic process; the exact cardiorespiratory pattern clinically detected depends on the complex interaction of patient, illness, time elapsed, and treatment provided.[4-9]

Because of its progressive nature, shock may be divided into phases: compensated, uncompensated, and irreversible.[4]

I) **Compensated or Early Shock**: Implies that vital organ function is maintained by intrinsic compensatory mechanisms such as venoconstriction, fluid shift from interstitial to intra-vascular space and arteriolar vasoconstriction.

The features are:

- Normal blood pressure
- Tachycardia.
- Narrow pulse pressure (as in hypovolemic shock) or wide pulse pressure (In septic shock).
- Signs of peripheral vasoconstriction evidenced by decreased skin temperature and impaired capillary refill >2secs.

• Signs of extracellular fluid loss like sunken eyes and anterior fontanelle, dry buccal mucosa and poor skin turgor may be present.

If shock is identified and vigorously treated at this stage, the syndrome may be successfully reversed.

II) **Decompensated Shock**: As the shock progresses to this state, the efficiency of the cardiovascular system is undermined, and microvascular perfusion becomes marginal despite compensatory adjustments. This phase has all the features of compensated shock and also has hypotension**.**

III) **Irreversible or terminal shock**: This phase implies damage to key organs of such magnitude that death occurs even if therapy returns cardiovascular parameters to normal levels.

By this stage, no matter what the initial classification of, a given shock state may have been, there are gross abnormalities in volume status, vascular tone, cardiac function, and cellular energetics and multiorgan failure. No currently measured parameter is sufficiently sensitive and specific to act as gold standard indicator of irreversible shock.[2]

Shock states may be classified into six functional categories:

• Hypovolemic

• Cardiogenic

• Obstructive

• Distributive

• Septic

• Miscellaneous**.**

Another functional category of shock, traumatic shock is also being used recently.

It is important to note that such tidy classifications imply a degree of precision that will be misleading when approaching an individual patient. Vicious cycles play a prominent role in most shock syndromes; any given patient, over time, may display features of any functional category.[5]

ETIOLOGIES OF THE DIFFERENT FUNCTIONAL CATEGORIES OF SHOCK

Hypovolemic shock: The causes of hypovolemic shock are listed below.[4]

1)*Whole blood loss*

i) **Hemorrhage - absolute loss**

a) External Bleeding

b) Internal Bleeding

c) Gastrointestinal

d) Intra-abdominal (spleen, liver)

e) Major vessel injury

f) Intracranial (in infants)

g) Fractures

ii) **Relative Loss**

a) Pharmacological (barbiturates, vasodilators)

b) Positive - pressure ventilation

c) Spinal cord injury

d) Sepsis

e) Anaphylaxis

2)*Plasma loss*

i)**Burns**

ii) **Capillary leak syndromes**

a) Inflammation sepsis

b) Anaphylaxis

iii) **Protein - losing syndromes**

a) Nephrosis

b) Intestinal disorders or obstruction

3)*Fluid and electrolyte loss*

a) Vomiting and diarrhea

b)Excessive diuretic use

c)Endocrine

Cardiogenic shock

The causes of cardiogenic shock are listed below.[11,12] Cardiac function can also be depressed in patients with shock that is not primarily due to a myocardial insult.

Myocardial dysfunction is frequently a late manifestation of shock of any etiology.[13]

1)*Heart rate abnormalities*

- Supraventricular tachycardia
- Ventricular dysarrhythmias
- Bradycardia

2)Cardiomyopathies / Carditis

- Infections
  - a) Sepsis
  - b) Myocarditis
- Hypoxic and ischemic events

a) Cardiac arrest

b) Prolonged shock

c) Head injury

d) Anomalous coronary artery

e) Excessive catecholamine states

3)Metabolic

a) Hypoglycemia

b) Hypocalcaemia

c) Acidosis

d) Thyroid disorders

e) Hypothermia

f) Glycogen storage disease

g) Carnitine deficiency

h) Mucopolysaccharidosis

4) Vascular, immunological

a) Kawasaki's disease

b) Polyarteritis nodosa

c) Systemic lupus erythematosus

d) Embolism

e) Acute rheumatic fever

5) Drug intoxication

6)Neuromuscular diseases

a) Duchenne's dystrophy

b) Friedreich's ataxia

7)Miscellaneous

a) Endocardial fibroelastosis

8)Congenital heart disease

9)Trauma

Obstructive shock

Causes of obstructive shock are acute pericardial tamponade, tension pneumothorax, pulmonary or systemic hypertension, and congenitally acquired outflow obstructions.[14]

Distributive shock

Distributive shock may be seen with anaphylaxis, spinal, or epidural anesthesia, disruption of the spinal cord, or inappropriate administration of vasodilatory medication.[14]

Septic shock

Septic shock can be caused by bacteria, virus, fungus, or protozoa.[15] Respiratory infections (37%) and primary bacteremia (25%) are the most common infections.[16]

According to American College of Critical Care Medicine guidelines the following are defined as:[17]

SIRS (a) (Systemic Inflammatory Response Syndrome)

The presence of at least two of the following four criteria,

One of which must be abnormal temperature or leukocyte count:

* Core temperature of >38.5°C or <36°C.

* Tachycardia, defined as a mean heart rate >2 SD above normal for age in the absence of external stimulus, chronic drugs, or painful stimuli; or otherwise unexplained persistent elevation over a 0.5- to 4-hr time period OR for children <1 yr old: Bradycardia, defined as a mean heart rate <10th percentile for age in the absence of external vagal stimulus, beta-blocker drugs, or congenital heart disease; or otherwise unexplained persistent depression over a 0.5-hr time period.

* Mean respiratory rate >2 SD above normal for age or mechanical ventilation for an acute process not related to underlying neuromuscular disease or the receipt of general anesthesia.

* Leukocyte count elevated or depressed for age (not secondary to chemotherapyinduced leucopenia) or >10% immature neutrophils.

Infection: A suspected or proven (by positive culture, tissue stain, or polymerase chain reaction test) infection caused by any pathogen OR a clinical syndrome associated with a high probability of infection. Evidence of infection includes positive findings on clinical exam, imaging, or laboratory tests (e.g., white blood cells in a normally sterile body fluid, perforated viscus, and chest radiograph consistent with pneumonia, petechial or purpuric rash, or purpura fulminans).

Sepsis: SIRS in the presence of or as a result of suspected or proven infection.

Severe sepsis: Sepsis plus one of the following: cardiovascular organ dysfunction OR acute respiratory distress syndrome OR two or more other organ dysfunctions.

Septic shock: Sepsis and cardiovascular organ dysfunction

ORGAN DYSFUNCTION CRITERIA[17]

Cardiovascular dysfunction

• Despite administration of isotonic intravenous fluid bolus > 40 mL/kg in 1 hr.

• Decrease in BP (hypotension) <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 mcg/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 >2 times upper limit of normal.

Oliguria: urine output <0.5 mL/kg/hr

Prolonged capillary refill: >3 secs

Core to peripheral temperature gap >3°C.

Respiratory

• PaO2/FiO2 <300 in absence of cyanotic heart / preexisting lung diseases OR

• PaCO2 >65 torr or 20 mm Hg over baseline PaCO2 OR

• Proven need or >50% FiO2 to maintain saturation >92% OR

• Need for non-elective invasive or noninvasive mechanical ventilation.

Neurologic

• Glasgow Coma Score <11 OR

• Acute change in mental status with a decrease in Glasgow Coma Score >3 points from abnormal baseline

Hematologic

• Platelet count: < 80,000/mm3 or a decline of 50% in platelet count from highest value recorded over the past 3 days (for chronic hematology/oncology patients).

OR

• International normalized ratio >2.

Renal

• Serum creatinine >2 times upper limit of normal for age or 2-fold increase in baseline creatinine.

Hepatic

• Total bilirubin >4 mg/dl (not applicable for newborn) OR

• ALT 2 times upper limit of normal for age (BP, blood pressure; ALT, alanine transaminase.

EPIDEMIOLOGY

Shock occurs in approximately 2% of all hospitalized children and adults in the United States (300-400, 000/yr).[1]

Hypovolemia is the most common cause of shock in children.[4] In health institutions in India up to a third of hospital admissions are diarrhea related and up to 17% of all deaths in indoor pediatric patients are gastroenteritis related.[18] The World Health Organization estimates that in developing countries 3 million children under the age of 5 years die of diarrhea each year, primarily because of hypovolemic shock, secondary to the vomiting and diarrhea that occurs with a variety of infectious agents.[19]

Pediatric severe sepsis per year in the United States is 0.56 cases per 1,000 population per year.[16] The incidence is highest in infants (5.16 per 1,000), fell dramatically in older children (0.20 per 1,000 in 10 to 14 year olds), and is15% higher in boys than in girls (0.60 versus 0.52 per 1,000, $p<0.001$). Hospital mortality was 10.3%, or 4,383 deaths nationally (6.2 per 100,000 population).[16] Septic shock is the most common cause of death in the medical and surgical intensive care units.[20]

There are approximately 1500 annual deaths from anaphylaxis in the United States.[21]

PATHOPHYSIOLOGY OF CIRCULATORY SHOCK – GENERAL OVERVIEW

Circulatory shock results in critical decreases in tissue perfusion that result in organ dysfunction.The initial response of the cardiovascular system to critical reduction to tissue perfusion is a complex set of reflexes that serve to maintain vascular tone and cardiac performance.

Increased sympathetic activity increases cardiac contractility and heart rate. Release of catecholamines, vasopressin, and angiotensin increases venular and arteriolar tone, augmenting central blood volume, venous return, and blood pressure.

Concomitantly, blood flow is preferentially redirected away from skeletal muscle, subcutaneous tissue, and splanchnic circulation to the brain and heart. Vasopressin and renin-angiotensin system also augment salt and water retention, thereby preserving intravascular blood volume.

As the shock state progresses, these mechanisms become less effective. Hypotension impairs coronary perfusion, thereby compromising cardiac output.

Further increases in peripheral vascular resistance adversely affect cardiac performance by increasing ventricular afterload. Tissue acidosis

and build up of other metabolites produce arteriole vasodilatation, worsening ongoing hypotension. When coupled with venular vasoconstriction, capillary hydrostatic pressures increase, with subsequent loss of intravascular volume.

In a group of patients, primarily with septic shock and some drug intoxications, the shock state is characterized by profound hypotension, unresponsive to endogenous and exogenous vasopressors. These patients have a marked hyperdynamic circulatory state and maldistribution of systemic blood flow resulting in tissue hypoperfusion. The deterioration to a hypodynamic state occurs as a terminal event in these patients.

PATHOPHYSIOLOGY OF THE FUNCTIONAL CATEGORIES OF SHOCK

1) **Hypovolemic Shock**

Hypovolemia is the most common cause of shock in infants and children. Hypovolemic shock is best defined as a sudden decrease in the intravascular blood volume relative to the vascular capacity to the extent that effective tissue perfusion cannot be maintained.[23]

Physiological mechanisms of the body compensate for the loss of intravascular fluid in children in the same way that they do in adults.[4, 7] Acute losses of 10% to 15% of the circulatory blood volume are well tolerated and in healthy children are easily compensated. Activation of peripheral and central baroreceptors produce an outpouring of catecholamines, and the resulting tachycardia and peripheral vasoconstriction are usually adequate to support the blood pressure with little or no evidence of hypotension. An acute loss of 25% or more of the circulating blood volume, however, frequently results in a clinically apparent hypovolemic state that requires immediate, aggressive management.[24]

The most reliable indicators of early, compensated hypovolemic shock in children are persistent tachycardia, cutaneous vasoconstriction, and diminution of the pulse pressure. The best clinical evidence of decreased tissue perfusion is skin mottling, prolonged capillary refill, and cold extremities. Systemic arterial blood pressure is frequently normal, the result of increased systemic vascular resistance.[4] Neurological status is normal or only minimally impaired. With continued loss of blood volume or with delayed or inadequate blood volume replacement, the intravascular fluid losses surpass the body's compensatory abilities, and decompensated phases appear. The pronounced systemic vasoconstriction and hypovolemia produce ischemia and stagnant hypoxia in the visceral and cutaneous circulations.[22] Altered cellular metabolism and function occur in these areas, resulting in damage to blood vessels, kidneys, liver, pancreas, and bowel. Stroke volume and cardiac output are decreased[4]. Patients are hypotensive, acidotic, lethargic or comatose, and oliguric or anuric. It is important to emphasize that arterial blood pressure falls only after compensations are exhausted, which may occur long after the precipitating event and after severe reduction in cardiac output.[25] Terminal

phases of hypovolemic shock are characterized by myocardial dysfunction and widespread cell death.

2) **Cardiogenic Shock**

Cardiogenic shock is the pathophysiological state in which an abnormality of cardiac function is responsible for the failure of the cardiovascular system to meet the metabolic needs of tissues.[4,26] The common denominator is depressed cardiac output, which in most instances is the result of decreased myocardial contractility. Cardiac function can also be depressed in patients with shock that is not primarily due to a myocardial insult. Myocardial dysfunction is frequently a late manifestation of shock of any etiology.

Although the cause of myocardial dysfunction in such patients, is not completely understood, the following mechanisms have been proposed:

(1) Specific toxic substances released during the course of shock that have a direct cardiac depressant effect.

(2) Myocardial edema.

(3) Adrenergic receptor dysfunction.

(4) Impaired sarcolemmal calcium flux.

(5) Reduced coronary blood flow resulting in impaired myocardial systolic and diastolic function.[13]

As opposed to hypovolemic shock; compensatory responses can have deleterious effects in patients with cardiogenic shock.[27,28] Compensatory responses are nonspecific and not precisely set, and in patients with cardiogenic shock they may contribute to the progression of shock by further depressing cardiac function. For example, as pump function deteriorates and cardiac output decreases, systemic vascular resistance increases in order to maintain circulatory stability. However, the increase in afterload adds to the heart's workload and further decreases pump function.[28] Therefore, in cardiogenic shock, a vicious cycle is established. Ventricular dysfunction is exacerbated by neurohumoral vasoconstrictive mechanisms, and vice versa. Because of the self-perpetuating cycle, compensated phases of cardiogenic shock may not be observed, and frequently only one cardiorespiratory pattern, in varying degrees of severity, is observed. The patients are tachycardic, hypotensive, diaphoretic, oliguric, and acidotic. Extremities are cool and mental status is altered. Hepatomegaly, jugular venous distention, rales, and peripheral edema may be observed.[4, 27] Cardiac output is depressed, and elevations in central venous pressure, pulmonary capillary wedge pressure, and systemic vascular resistance are observed.

3) **Obstructive Shock**

Obstructive shock is caused by the inability to produce adequate cardiac output despite normal intravascular volume and myocardial function. Causative factors may be within the pulmonary circulation or the systemic circulation or associated with the heart itself. Examples of obstructive shock include acute pericardial tamponade, tension pneumothorax, pulmonary or systemic hypertension, and congenitally acquired outflow obstructions. Recognition of the characteristic features of these syndromes is essential, as most of the causes are treatable, provided the diagnosis is made early.[14]

4) **Distributive Shock**

Distributive shock results from maldistribution of blood flow to the tissue.

Abnormalities in the distribution of blood flow may result in profound inadequacies in tissue oxygenation, even in the face of a normal or high cardiac output. Such maldistribution of flow generally results from widespread abnormalities in vasomotor tone. Distributive shock may be seen with anaphylaxis, spinal, or epidural anesthesia, disruption of the spinal cord, or inappropriate administration of vasodilatory medication.[14]

5) **SEPTIC SHOCK**

Septic shock is the most complex and controversial type of shock and merits independent classification. Septic shock often is the combination of multiple problems, including hypovolemia, maldistribution of blood flow, myocardial depression, and multiple metabolic and endocrinological problems.[29,30]

This form of shock is caused by the systemic response to a severe infection. Gram-positive and -negative bacteria, viruses, fungi, rickettsiae, and protozoa have all been reported to produce the clinical picture of septic shock, and the **overall response is generally independent of the specific type of invading organism.** The clinical findings in septic shock are a consequence of the combination of metabolic and circulatory derangements driven by the systemic infection and the release of toxic components of the infectious organisms, e.g., the endotoxin of gram-negative bacteria or the exotoxins and enterotoxins of gram-positive bacteria. Organism toxins lead to the release of cytokines, including IL-1.IL-6, IL-8, IL-12, INF gamma, G-CSF and TNF-a, from tissue macrophages. Tissue factor expression and fibrin deposition are increased, and disseminated intravascular coagulation may develop. The inducible

form of NO synthase is stimulated, and NO, a powerful vasodilator, is released. Hemodynamic changes in septic shock occur in two characteristic patterns: early, or hyperdynamic, and late, or hypodynamic, septic shock.

Hyperdynamic Response: In hyperdynamic septic shock, tachycardia is present, the cardiac output is normal, and the systemic vascular resistance is reduced while the pulmonary vascular resistance is elevated. The extremities are usually warm. However, splanchnic vasoconstriction with decreased visceral flow is present. The venous capacitance is increased, which decreases venous return. With volume expansion cardiac output becomes supranormal. Myocardial contractility is depressed in septic shock by mediators including NO, IL-1, and/or TNF-a. Inflammatory mediator-induced processes include increased capillary permeability and continued loss of intravascular volume.

In septic shock, in contrast to other types of shock, total oxygen delivery may be increased while oxygen extraction is reduced due to maldistribution of microcirculatory perfusion and impaired utilization. In this setting the presence of normal mixed venous oxygen saturation is not indicative of adequate peripheral perfusion, and even though the cardiac

output may be elevated, it is still inadequate to meet the total metabolic needs.

The toxicity of the infectious agents and their byproducts and the subsequent metabolic dysfunction drive the progressive deterioration of cellular and organ function. Acute respiratory distress syndrome, thrombocytopenia, and neutropenia are common complications.

Hypodynamic Response: As sepsis progresses, vasoconstriction occurs and the cardiac output declines. The patient usually becomes markedly tachypneic, febrile, diaphoretic, and obtunded, with cool, mottled, and often-cyanotic extremities. Oliguria, renal failure, and hypothermia develop; there may be striking increases in serum lactate.[15]

6) **Traumatic Shock**

Shock following trauma is, in large measure, due to hypovolemia. However, even when hemorrhage has been controlled, patients can continue to suffer loss of plasma volume into the interstitium of injured tissues. These fluid losses are compounded by injury-induced inflammatory responses, which contribute to the secondary microcirculatory injury. This causes secondary tissue injury and maldistribution of blood flow, intensifying tissue ischemia and leading to

multiple organ system failure. Trauma to the heart, chest, or head can also contribute to the shock. For example, pericardial tamponade or tension pneumothorax impairs ventricular filling, while myocardial contusion depresses myocardial contractility.[15]

The detection of altered organ function in the acutely ill patient constitutes multiple organ dysfunction syndromes (two or more organ involvement). The terminology dysfunction identifies this process as a phenomenon in which organ function is not capable of maintaining homeostasis. This process, which may be absolute or relative, can be more readily identified as a continuum of change over time.

COMPLICATIONS OF SHOCK

Respiratory failure is a frequent complication in shock and may be due to failure of the ventilator pump, i.e., respiratory muscle fatigue or deterioration of lung function, i.e., respiratory distress syndrome.[4,31] For these reasons, increased inspired oxygen is essential in all children with shock. In order to ensure the airway, provide relief from respiratory muscle fatigue, and facilitate provision of positive airway pressure, early tracheal intubation should be considered.[4, 10]

Progressive azotemia, with or without oliguria, may develop in association with any of the shock syndromes. The shock-related renal failure syndromes are a continuum from acute prerenal failure, through classic acute tubular necrosis, to the extreme of cortical necrosis. Although the precise mechanisms involved in the production of renal failure are unclear, diminished renal perfusion because of persistent vasospasm with reduced glomerular filtration rate, enhanced distal exchange site activity secondary to increased aldosterone production, and increased free water absorption under the influence of elevated antidiuretic hormone activity all seem to be operative[10]. High output renal failure may occur in shock states, without any previous episodes of oliguria. This may falsely suggest adequate renal perfusion and adequate prerenal augmentation at a time when the patient's intravascular volume is, in fact, being depleted.[4, 32]

Coagulation abnormalities (e.g., disseminated intravascular coagulation) probably occur to some extent in all forms of shock. Monitoring of prothrombin time, partial thromboplastin time, and platelet count and observation for excessive bleeding are essential.[32]

Hepatic dysfunction occurs in varying degrees in most shock states.

Gastrointestinal disturbances after hypoperfusion and stress include bleeding and ileus. Ileus may result from electrolyte abnormalities and may lead to abdominal distention with respiratory compromise.

Multiple endocrinological problems may arise and complicate the management of children in shock. Included in these are problems with fluid, electrolytes, and mineral balance. Severe abnormalities of calcium homeostasis can occur in the course of any acute hemodynamic deterioration. Marked decreases in serum ionized calcium levels have been reported in conditions associated with inadequate tissue perfusion, regardless of etiology.[14]

DIAGNOSIS OF SHOCK

Shock is a clinical diagnosis.[2] The history and the clinical evaluations will facilitate early etiologic classification of shock, and help in directing appropriate treatment.[33] This is the simplest and most rapid means for detecting the state of inadequate perfusion, determining which tissues are compromised, and gauging the efficacy of therapy.[2]

HISTORICAL INFORMATION AND CLINICAL SIGNS IN DIFFERENT SHOCK FORMS

	HYPOVOLEMIC SHOCK	CARDIOGENIC SHOCK	DISTRIBUTIVE SHOCK
History	Trauma, vomiting, diarrhea	Congenital heart disease, past cardiac surgery, refusal of feeds and respiratory distress	Fever, lethargy, poor feeding, irritability and abnormal skin colour.
Heart rate	Increased	Increased	Increased
Chest x-ray			
Heart size	Small	Large	Small
Lungs	Clear	Wet	Clear (in the early stage)
Gallop rhythm	Not present	Present	Not present
Capillary refill time	Prolonged	Prolonged	Normal (in the early stage)

STAGES OF SEPTIC SHOCK[33]

Early stage (hyperdynamic)	Late stage (cardiogenic)
1. Hyperthermia	1. Hypothermia
2. Tachycardia	2. Tachycardia
3. Tachypnea	3. Bradypnea
4. Warm extremities	4.Cold mottled extremities
5. Bounding pulse	5. Weak, thready pulse
6. Normal capillary refill	6. Prolonged capillary refill
7. Normotensive/hypertensive	7. Hypotensive
8. Hypoxia	8. Hypoxia
9. Polyuria	9. Oliguria/anuria
10. Increased cardiac output	10. Decreased cardiac output
11. Decreased SVR	11. Increased SVR
12. Normal CNS	12. Obtunded, comatose
13. Respiratory alkalosis	13. Metabolic acidosis
14. Hyperglycemia	14. Hypoglycemia
15. Normal coagulation	15. Disseminated intravascular coagulopathy

CNS → Central Nervous System, SVR → Systemic Vascular Resistance

Further assessment of the severity and cause of shock states is greatly assisted by laboratory investigations. Routine laboratory tests such as serum electrolytes, serum calcium blood cell counts, platelet counts, and hematocrit are obviously necessary to delineate the extent of metabolic disturbance. Probably the most valuable investigation is the arterial blood gas analysis.

Arterial oxygen content and carbon dioxide tension aid in the adequacy of ventilatory function that is frequently impaired in shock. In addition pH and base deficit determination serves as one of the most readily available methods of quantifying tissue hypoperfusion.[2]

Management of shock[17]

In 2003, critical care and infectious disease experts representing 11 international organizations developed management guidelines for other supportive therapies in sepsis that would be of practical use for the bedside clinician, under the auspices of the Surviving Sepsis Campaign, an international effort to increase awareness and to improve outcome in severe sepsis. Practical application of this information in Indian set up in a

child with septic shock will be discussed. In 1992, ACCP/SCCM consensus guidelines for definitions of sepsis were published by Bone et al.

MONITORING OF SHOCK

The most effective and sensitive physiologic monitoring available is the frequent, repeated examination of the child by a competent, careful observer. Observations for alterations in peripheral perfusion by examining capillary refill time and core –peripheral temperature gradient, color, presence of cyanosis, characteristics of the pulse, blood pressure, respiratory pattern, and level of consciousness are absolutely essential in the continuous and ongoing monitoring of children with shock. Careful nursing observation of vital signs and activity of the child and clear, concise display of these data from the central core of information from which the child's therapy is determined.[2]

Minimal monitoring of the child in shock or at risk for shock should include continuous electrocardiographic monitoring, frequent blood pressure and temperature measurements, and measurement of blood glucose in younger infants.[34]

Other variables that have been used to monitor patients, guide therapy, and predict outcome include mean arterial pressure, central venous pressure, Swan-Ganz catheter placement, mixed venous oxygen saturation, oxygen delivery and consumption, gastric mucosal pH, blood lactate level, and echocardiography.[35]

Early goal directed therapy helps keep the cost and duration of hospital stay to a minimum.

Need for early intubation and ventilation

Due to low functional residual capacity, young infants and neonates with severe sepsis may require early intubation. Unfortunately no objective clinical criteria specific to pediatric septic shock for timing of endotracheal intubation (other than the standard indications, which include shock) exist in literature. Therefore it is reasonable to consider endotracheal intubation when shock is persistent even after a volume resuscitation of >40-60 ml/kg. Children with sepsis requiring aggressive fluid resuscitation frequently have worsening tachypnea and increasing oxygen requirement clinically depicting early acute respiratory distress syndrome (ARDS).These patients will require early intubation and mechanical ventilation. The principles of lung- protective strategies (low

tidal volumes and permissive hypercapnea) are applied to children as they are to adults. In premature infants, additional attention is paid to avoiding hyperoxemia to prevent retinopathy.

Fluid Resuscitation

Intravenous access for fluid resuscitation and inotrope/vasopressor infusion is more difficult to attain in children than in adults. The American Heart Association has well established Pediatric advanced life support (PALS) guidelines for emergency establishment of intravascular support including intraosseous access. On the basis of many studies, it is accepted that aggressive fluid resuscitation with crystalloids or colloids is of fundamental importance to survival of septic shock in children.

There is only one randomized, controlled trial comparing the use of colloid with crystalloid resuscitation (dextran, gelatin, lactated Ringers, or saline) in children with dengue shock. All these children survived, regardless of the fluid used, but the longest time to recovery from shock occurred in children who received lactated Ringers. Among patients with the narrowest pulse pressure, there was a suggestion that colloids were more effective than crystalloids in restoring normal pulse pressure. Fluid infusion is best initiated with boluses of 20mL/kg over 5-10 mins, titrated

to clinical monitors of cardiac output, including heart rate, urine output, capillary refill, and level of consciousness.

A 60 ml syringe filled with fluid drawn via the fluid bag with a three-way connection can be conveniently used to push fluid boluses in the absence of a volumetric pump. Children normally have a lower blood pressure than adults and can prevent reduction in blood pressure by vasoconstriction and increasing heart rate. Therefore, blood pressure by itself is not a reliable endpoint for assessing the adequacy of resuscitation.

However, once hypotension occurs, cardiovascular collapse may soon follow.

Hepatomegaly occurs in children who are fluid overloaded and can be a helpful sign of the adequacy of fluid resuscitation. Other practical ways to assess fluid overload are jugular venous distension, heart size and pulmonary congestion on chest x ray. Gold standard still remains the measurement of a central venous pressure. Large fluid deficits typically exist, and initial volume resuscitation usually requires 40-60 ml/kg but can be much higher.

Vasopressors / Inotropes

Should only be used after appropriate volume resuscitation. Children with severe sepsis present with low cardiac output and high systemic vascular resistance (cold shock, more common scenario), high cardiac output and low systemic vascular resistance, or low cardiac output and low systemic vascular resistance shock.

Early inotropic support should be started in the case of fluid refractory shock or a life threatening hypotension when fluid bolus has been initiated. Dopamine is the first choice of support for the pediatric patient with hypotension refractory to fluid resuscitation. The choice of vasoactive agent is determined by the clinical examination. Dopamine-refractory shock may reverse with epinephrine (adrenaline) or norepinephrine (noradrenaline) infusion.

Pediatric patients with low cardiac output states may benefit from use of dobutamine. The use of vasodilators can reverse shock in pediatric patients who remain hemodynamically unstable with a high systemic vascular resistance state despite fluid resuscitation and implementation of inotropic support. Nitrosovasodilators with a very short half-life (nitroprusside or nitroglycerin) are used as first-line therapy for children

with epinephrine-resistant low cardiac output and elevated systemic vascular-resistance shock.

Inhaled nitric oxide reduced extracorporeal membrane oxygenation use when given to term neonates with persistent pulmonary artery hypertension of the newborn and sepsis in a randomized, controlled trial. When pediatric patients remain in a normotensive low cardiac output and high vascular resistance state, despite epinephrine and nitrosovasodilator therapy, then the use of a phosphodiesterase inhibitor should be strongly considered, such as milrinone. Vasopressin therapy should be considered in warm shock unresponsive to fluid and norepinephrine.

Early antibiotics

After appropriate cultures are taken early use of broad spectrum systemic antimicrobial therapy based on clinical suspicion is reasonable although no randomized studies exist in children. Adult data supports use early appropriate antibiotics to impact favorably on morbidity from septic shock.

Therapeutic end points

Therapeutic endpoints are capillary refill of <2 secs, normal pulses with no differential between peripheral and central pulses, warm limbs,

urine output of >1 ml/kg/hr, normal mental status, decreased lactate, and increased base deficit and superior venacava or mixed venous oxygen saturation of >70%. When employing measurements to assist in identifying acceptable cardiac output in children with systemic arterial hypoxemia such as cyanotic congenital heart disease or severe pulmonary disease, arterial-venous oxygen content difference is a better marker than mixed venous hemoglobin saturation with oxygen. Optimizing preload optimizes cardiac index.

As noted above, blood pressure by itself is not a reliable endpoint for resuscitation. Rarely, if a pulmonary artery catheter is utilized, therapeutic endpoints are cardiac index of >3.3 and <6.0 L/m/meter sq with normal perfusion pressure (mean arterial pressure-central venous pressure) for age. Use of pulmonary artery catheter has declined over the years due to no well-demonstrated therapeutic benefit in patients with septic shock.

Electrolyte balance

An attempt should be made to check and correct common electrolyte problems related to sodium (hyponatremia), potassium and ionized calcium (ionized hypocalcemia).

Steroids

Hydrocortisone therapy should be reserved for use in children with catecholamine resistance and suspected or proven adrenal insufficiency. Patients at risk include children with severe septic shock and purpura, children who have previously received steroid therapies for chronic illness, and children with pituitary or adrenal abnormalities.

Dose recommendations vary from 1-2 mg/kg for stress coverage (based on clinical diagnosis of adrenal insufficiency) to 50 mg/kg for empirical therapy of shock followed by the same dose as a 24-hr infusion. Thus dose of steroids remains controversial.

PROGNOSTIC FACTORS IN SHOCK

Overall mortality of shock is 20-50%.[1] The mortality depends on the underlying etiology. Septic shock is associated with high mortality whereas hypovolemic shock is associated with least mortality. Mortality rates in septic shock in adults ranges from 40-60%.[36] In children it varies from 10 to 82%.[16,37-39] In septic shock, outcome in children is markedly better than in adults (9% in children compared with 28% mortality in adults).[40]

In a retrospective study of the risk factors determining outcome of nontraumatic patients with shock in the pediatric emergency service of a University Hospital Chang P et al have identified 22 patients with the diagnosis of shock which included 11 with septic shock (50%), 7 with hypovolemic shock (32%) and 4 with cardiogenic shock (18%). Their age ranged from 2 months to 19 years old. Gram-negative bacterial sepsis (6/11, 55%), dilated cardiomyopathy (2/4, 50%) and acute gastroenteritis (7/7, 100%) were the most frequent causes of septic, cardiogenic and hypovolemic shock, respectively. In total, 12 patients (55%) died. The mortality rate was high in septic shock (9/11, 82%) and cardiogenic shock patients (3/4, 75%), but low in hypovolemic shock patients (0/7, 0%). The

risk factors of poor outcome in patients with shock included thrombocytopenia, prolonged prothrombin time and partial thromboplastin time. Patients with leukopenia, a higher level of C-reactive protein, or under 2 years of age tended to have poor outcome.[39]

Multiple organ systems failure increases the probability of death (one organ system involved=25%; two organ systems=60% three or more organ systems=>85%).[1] When the central nervous system is injured, this often becomes the limiting factor that prevents survival. Also ultimate outcome in severe shock states is often affected by hepatic dysfunction.[14] Banks and colleagues found that clinical jaundice was apparent in 63 percent of their patients with septic shock, that it was more common in non-survivors than survivors, and that the degree of biochemical liver abnormalities was related to the duration of shock.[41]

The French ICU Group for Severe Sepsis have concluded that the major determinants of both early (< 3 days) and secondary deaths in adults with severe sepsis were the Simplified Acute Physiology Score II (SAPS II) and the number of acute organ system failures. Other risk factors for early death included a low arterial blood pH (<7.33) and shock, whereas secondary deaths were associated with the admission category, a

rapidly or ultimately fatal underlying disease, a preexisting liver or cardiovascular insufficiency, hypothermia, thrombocytopenia, and multiple sources of infection. In patients with documented sepsis, bacteremia was associated with early mortality.[42]

In case of septic shock neutropenia, hypothermia, and encephalopathy are associated with poor outcome. Most patients who do not recover initially die later. The correct choice of antibiotic has consistently been associated with improved outcomes from septic shock.[19]

Pollock and associates, in their study in pediatric patients with septic shock have shown that the outcome is improved in patients with increased cardiac output, elevated oxygen consumption and elevated oxygen extraction without significant pulmonary disease. On the other hand, low body temperature (< 37 degrees. centigrade), pulmonary disease, low cardiac index (< 3.3 I/min/m2) and decreased oxygen utilization are all poor prognostic indicators in shock.[43]

They suggested that the following changes are associated with poor prognosis

(a) cardiac index values that either do not increase appreciably in the middle and latter periods or which increase more than 100% above

control values; (b) mean arterial pressures below 50 mm hg in the early period; (c) sustained increase in the pulmonary vascular resistance of more than 500 dyne.sec/cm5/sq m in the early and middle period; (d) arterial PCO2 more than 50 mmHg; (e) pH below 7.3 or above 7.6; (f) oxygen consumption below 120 ml/min/sqm in the early period and above 250 ml/min/sq min in the late period.

Additional well known information from the literature and common clinical experience suggest that the following, if prolonged, may also indicate a poor prognosis: (a) urine outputs below 20 ml/hr; (b) CVPs of more than 20 cm H2O; (c) heart rates over 150 and under 70 beats per minute; (d) arrhythmias; (e) hematocrit vales below 25%; (f) markedly increased work of respiration;(g) decreased ventilatory compliance;(h) increased pulmonary venous admixture (shunting); (i) increased ventilatory dead space; and (j) increased plasma lactate levels and "excess lactate".[45]

Heart rate is an early predictor of prognosis in septic shock. Parker MM et al have done a study on forty-eight adult patients with septic shock, of whom 19 (40%) were survivors and 29 non survivors. At the initial evaluation, both survivors and non survivors demonstrated an

elevated cardiac index (CI), low systemic vascular resistance index (SVRI), and normal stroke volume index. However, only an initial heart rate (HR) less than 106 beat/min significantly predicted survival. Twenty-four hours after the onset of shock, an HR less than 95 beats/min and an SVRI greater than 1529 dyne.sec/cm5.m2 predicted survival. Comparing the hemodynamic profiles from the initial to the 24 h time point, a decrease in HR greater than 18 beats/min or a decrease in CI greater than 0.5 L/min.m2 predicted survival.[52]

To identify early prognostic markers of septic shock among catheterization derived hemodynamic and metabolic data a prospective cohort study was done at a medical intensive care unit in a university hospital. Thirty-two consecutive adult patients with septic shock, separated into two groups according to short-term (10-day) evolution:
18 acute survivors and 14 fatalities. Usual hemodynamic and metabolic variables were measured at the onset of shock, i.e., when the catheter was inserted (TO), and 24 h later (T24). The values collected for each group at TO and T24 and their 24-h changes were compared. On admission, no difference was found between acute survivors and eventual fatalities. After 24 h, fatalities presented with significantly lower mean arterial

pressure, left ventricular stroke work index and higher lactate levels than acute survivors.

Moreover, the 24-h changes of lactate and blood pressure were also of prognostic value.

Oxygen delivery and oxygen consumption did not differ statistically between the two groups. At T24, a mean arterial pressure of less than 85 mmHg and a lactate level equal to or greater than 3.5mmo/l1 were independently associated with poor survival (37.5% and 30.7%, respectively). Day 10 survival was only 12.5% when both criteria were present at T24 Changes in mean arterial pressure and arterial blood lactate within the first 24 h of treatment are strong prognostic indicators of short-term survival in patients with septic shock. After 24 hour of treatment, maintenance of a mean blood pressure equal to or greater than 85 mmHg correlates with survival at day 10. This data suggest that early reductions in both cardiac function and vascular tone play a determining role in the hypotension observed in fatalities. Persistence of hyperlactatemia in hypotensive patients is associated with poor survival. Blood pressure and lactate level are simple bedside parameters that can enable the clinician to identify patients with a high risk of mortality.[54]

Hyperlactatemia is an important prognostic marker in shock states. In a study by Hatherill M et al. hyperlactatemia was predictive of a poor outcome. There was no association between the magnitude of metabolic acidosis, quantified by the base excess, and mortality in children with shock.[55] Suistomaa M et al, observed that hyperlactatemia persisting more than 6 hour and simultaneous elevation of lactate/pyruvate ratio are associated with increased mortality in critically ill patients.[56] In the study done by Duke TO et al, blood lactate level was the earliest predictor of outcome in children with sepsis. The mean arterial pressure distinguished survivors from non-survivors at 24 and 48 hours. The base deficit and heart rate did not identify non-survivors from survivors at any time in the first 48 h. They also concluded that in children with sepsis, gastric tonometry added little to the clinical information that could be derived more simply by other means.[57] After surgery for complex congenital heart disease in children initial lactate concentrations were a poor predictor of mortality .However, elevated serum lactate levels indicated postoperative complications. In the same study use of base deficit was of no value in predicting mortality.[58]

Core-peripheral temperature gradient can also be used to predict outcome, although it does not relate to cardiac output or systemic vascular resistance.[59] Failure to increase toe temperature after heart surgery has been associated with an increased risk for death in children after heart surgery.[60] With the onset of shock, toe temperature can approach an ambient level (22-25°C); in one study, patients recovering from shock showed widening of toe: ambient gradient of more than 4°C, whereas in those who died, the toe: ambient gradient remained 1 to 2c.[61]

In pediatric septic shock, the admission PCT, like TNF and IL-10, is related to the severity of organ failure and mortality and a fall in PCT after 24 hrs of treatment may have favorable prognostic significance.[62]

METHODOLOGY

This was a prospective observational study of 81 consecutive children's admitted with shock in the pediatric ward of Government Rajaji Hospital, Institute of Child Health & Research Centre ,Madurai , over a period of 12 months from October 2009 to September 2010.

INCLUSION CRITERIA

• Children more than 1 month and upto 12 years with a clinical diagnosis of shock.

EXCLUSION CRITERIA

• Neonates

• Children with traumatic shock (hypovolemic shock due to trauma)

• Children who die within one hour after admission and patients in terminal state of cardiorespiratory failure.

81 consecutive cases admitted with a clinical diagnosis of shock fulfilling the below criteria were taken and their clinical and investigational parameters were studied and compared between survivors and non-survivors.

Consent was obtained from the parents/caregivers. Ethical committee clearance was taken.

WORKING CRITERIA:

Shock was defined as a clinical state in which the recorded blood pressure was <2 standard deviations below the mean for age and/or a state in which **at least three** of the following criteria for decreased perfusion were identified:

1) Decreased peripheral pulses

2) Mottled or cool extremities

3) Tachycardia (heart rate> 180 beats per minute for infants and> 160 beats per minute for children); or

4) Urine output <1 ml/kg/h, if <30 kg and <0.5 ml/kg if >30 kg.

5) Capillary refill time.

Hypovolemic shock was diagnosed when there was history of fluid loss like vomiting, diarrhea, loss of blood etc and physical findings of dehydration and shock.

Cardiogenic shock was identified when there was preexisting heart disease or when there were known risk factors to cause myocardial damage like scorpion sting and the findings also pointing towards a primary cardiac involvement and concomitantly having features of shock mentioned above.

Septic shock was diagnosed when there was a focus of infection like meningitis, encephalitis, or pneumonia proven by clinical features and appropriate investigations and also having features of hemodynamic compromise.

Anaphylactic shock was said to be there when there was sudden cardiovascular collapse following exposure to an inciting agent.

Dengue shock was identified when child has fever (2-7 days) and hemorrhagic features evidence by one /more of following:

1. Petechiae / purpura / ecchymosis
2. Positive tourniquet test
3. Bleeding from GIT (Hematemesis / Melena)
4. Thrombocytopenia ≤ 1 lakh cells / cu.mm
5. Plasma leakage (Ascites, pleural effusion, > 20% rise in hematocrit) plus signs of circulatory failure.

Once the patient was presented to the emergency room the relevant history was taken quickly while instituting appropriate treatment.

The patients were monitored for the following parameters:

1. Heart rate
2. Blood pressure

3. Respiratory rate
4. Capillary Refill Time (CRT)
5. Core-peripheral temperature gradient (C-PTG)
6. Glasgow Coma Scale (GCS)
7. Oxygen saturation (SpO2) and
8. Urine output
9. Peripheral pulses

These parameters were recorded periodically from the time of presentation and during the hospital stay. The readings at 0, 12, 24 and 48 hours after admission were analyzed and investigations done at admission were also analyzed. Consciousness was assessed using modified GCS for infants and children. Heart rate was obtained from the multichannel monitoring. Also the pulse was felt and its character assessed, as well as blood pressure recording was obtained non-invasively. Respiratory rate was counted and recorded.

Capillary refill time was recorded in the following manner: the upper limb was raised slightly above the level of the heart and firm pressure was applied by the clinician's index finger and thumb to the distal phalanx of the patient's index finger for five seconds.

The finger was then released and the time taken for the palmar pulp to return to its previous color was recorded. Times were measured to the nearest second by a wristwatch.

Core temperature was measured rectally, and peripheral temperature taken on the distal aspect that was not overtly ischemic. SpO2 was measured by pulse oximetry.

All the patients were catheterized and the urine output was measured.

Therapy was given based on existing protocols in the institute. For hypovolemic shock fluid boluses were given to restore the blood pressure and then subsequently dehydration assessed and corrected. In children with cardiogenic shock Dobutamine and vasodilator, were used.

Septic shock cases were treated with initial 3 boluses of crystalloids and then dopamine started if they had persistent shock. If there was no response to maximum dose of dopamine (15 μg/kg /min), adrenaline infusion was started. In anaphylactic shock cases, adrenaline infusion started along with volume expansion.

Dengue shock cases were treated with

5% DNS boluses - 20ml /kg/hr followed by 10ml /kg/hr infusion.

If patient improves - IV fluids gradually reduced to 6ml/kg/hr and then to 3ml/kg/hr upto 48 hrs.

If patient doesn't improve - Fresh whole blood transfusion given at 10ml/kg/hr and fluid therapy continued at 10ml/kg/hr waiting for response. Platelet transfusion were given when platelets <10,000 /cu.mm.

The outcome measure was ultimate survival or death.

Statistical analysis were done using SPSS software in the computer.

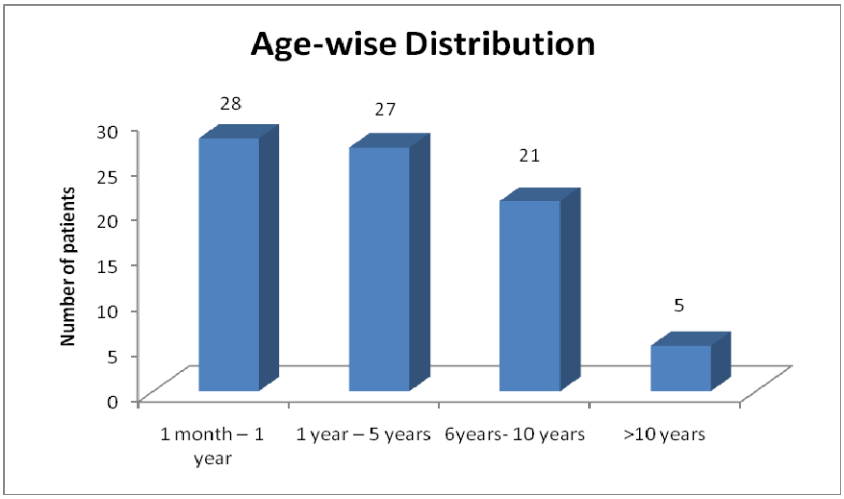
OBSERVATIONS AND RESULTS

Study design: A prospective clinical study of 81 patients with shock was undertaken.

Table 1: Age distribution of patients studied

AGE	NUMBER	PERCENTAGE
1 month – 1 year	28	34.56 %
1 year – 5 years	27	33.33 %
6years- 10 years	21	25.92 %
>10 years	5	6.1 %
TOTAL	81	100 %

Figure -1

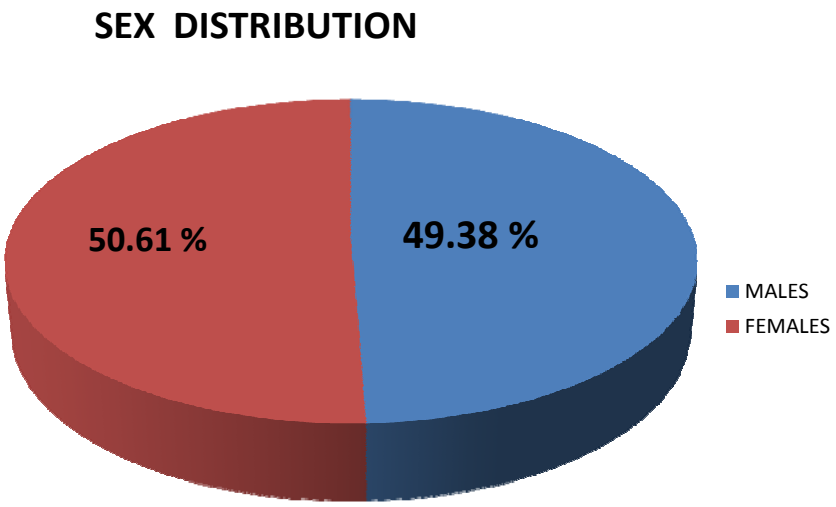


Most common age group is between 1 months- 5 years which constitutes 67.89%.

Table 2: Sex distribution

SEX	NUMBER	PERCENTAGE
Male	**40**	**49.38 %**
Female	**41**	**50.61 %**
Total	**81**	**100 %**

Figure 2

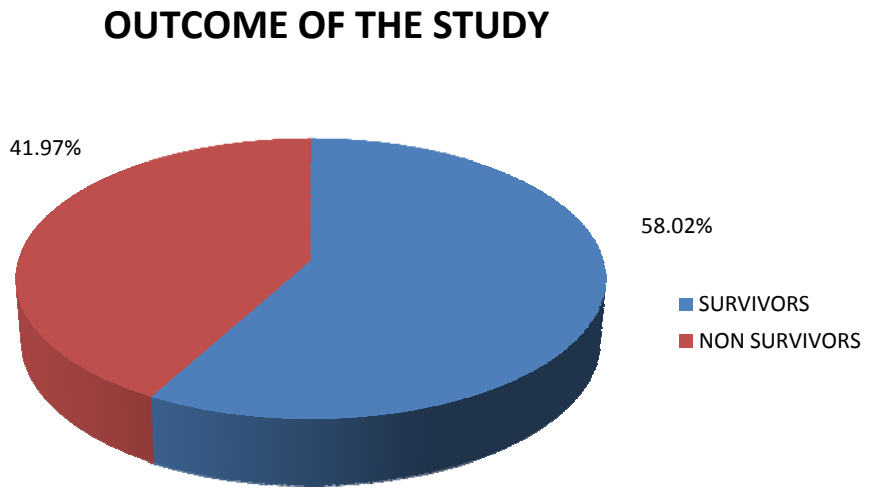


Out of 81 cases, 49.38% were males and 50.61% were females.

Table 3: Outcome of the study

OUTCOME	NUMBER	PERCENTAGE
SURVIVORS	47	58.02 %
NON-SURVIVORS	34	41.97 %
TOTAL	**81**	**100 %**

Figure 3

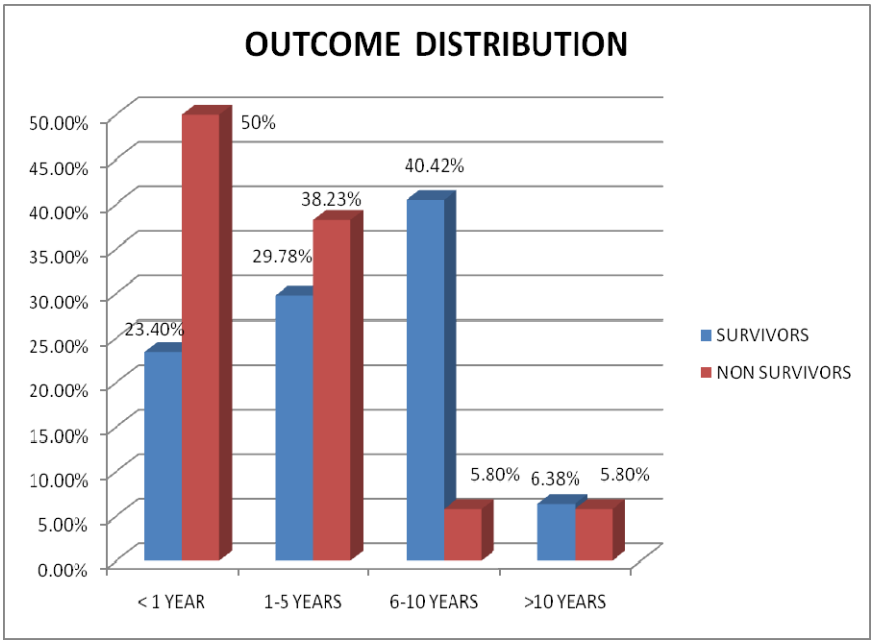


Out of 81 cases ,58.02% survived and 41.97% were non –survivors.

Table 4: Distribution of outcome according to age

AGE	SURVIVORS (n = 47)		NON- SURVIVORS (n =34)	
	NO	%	NO	%
Upto 1 year	11	23.4 %	17	50 %
1 year -5 years	14	29.78 %	13	38.23 %
6 years-10 years	19	40.42 %	2	5.8 %
>10 years	3	6.38 %	2	5.8 %

Figure 4
Distribution of outcome according to age

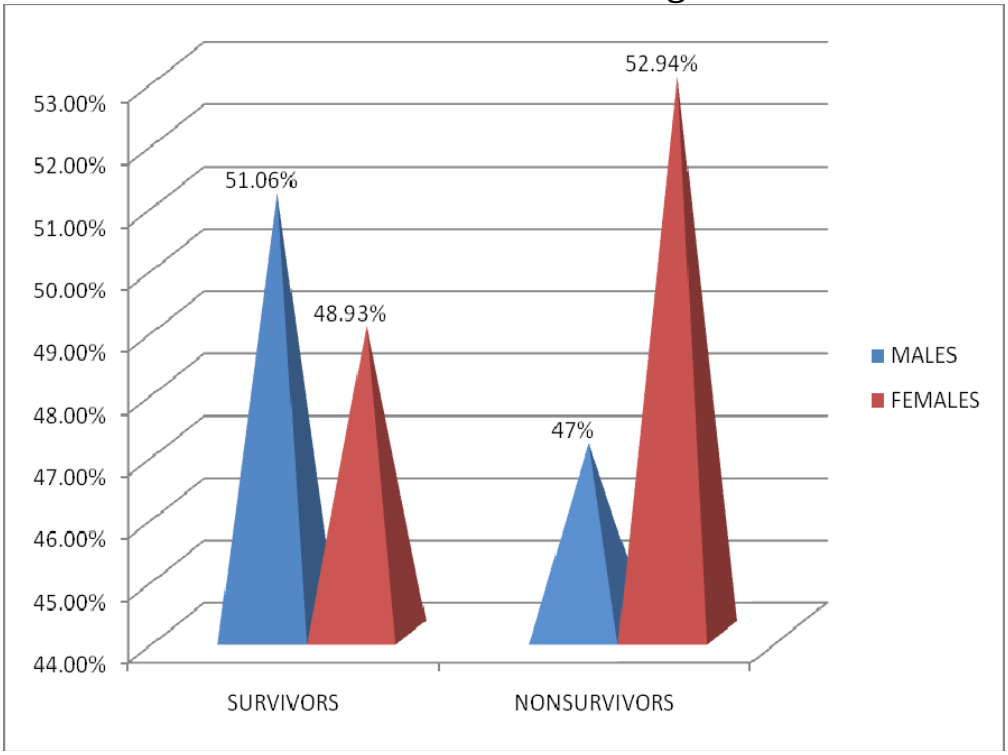


Mortality rate is higher (50%) among infants < 1 year, while survival rate is higher among children among 6 – 10 years age group.

Table 5: Distribution of outcome according to sex

SEX	SURVIVORS (n = 47)		NON-SURVIVORS (n=34)	
	NO	%	NO	%
MALE	24	51.06 %	16	47.05 %
FEMALE	23	48.93 %	18	52.94 %

Figure 5
Distribution of outcome according to sex

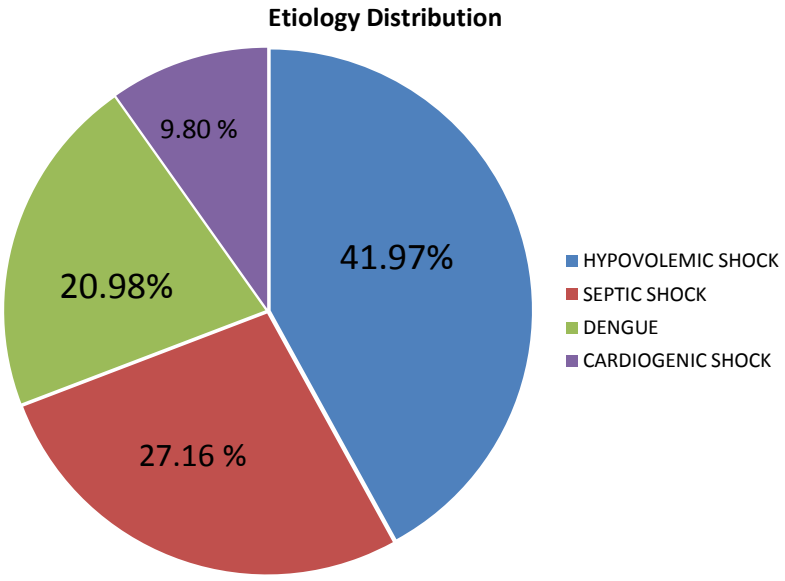


Out of 34 non survivors, 47.05 % were males and 52.94 % were females.

Table 6a: Etiology distribution

ETIOLOGY	NUMBER	PERCENTAGE
Hypovolemic shock	34	41.97 %
Septic shock	22	27.16 %
Cardiogenic shock	8	9.8 %
Dengue shock	17	20.98 %
Total cases	**81**	**100 %**

Figure 6



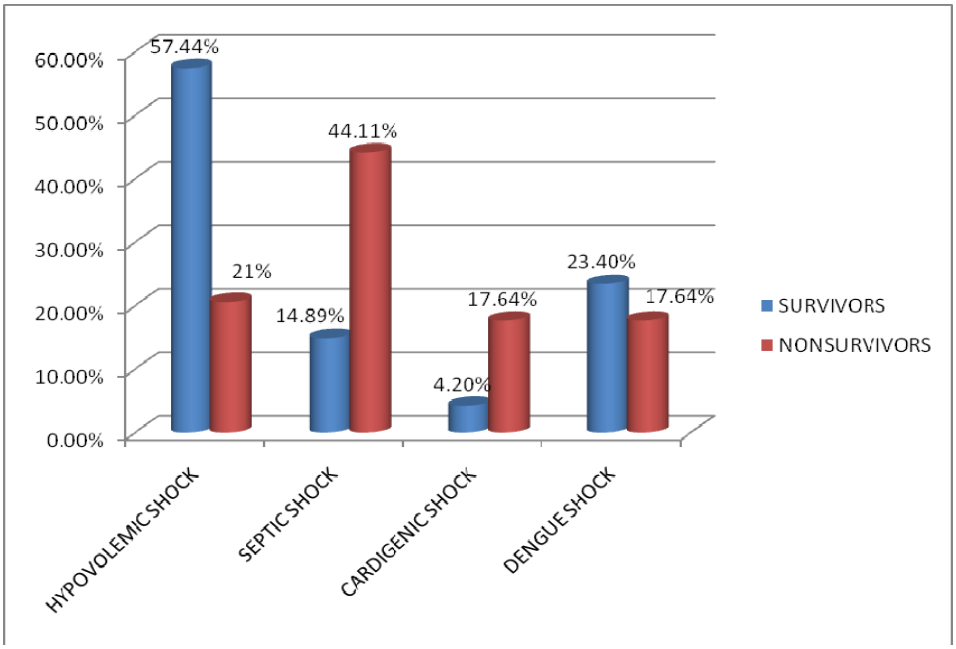
Out of 81 cases, Hypovlemic shock is most common 41.97% and cardiogenic shock is least common (9.80%).

Table 7: Distribution of outcome according to etiology

ETIOLOGY	SURVIVORS (n = 47)		NON SURVIVORS (n =34)	
	NO	%	NO	%
Hypovolemic shock	27	57.44 %	7	20.58 %
Septic shock	7	14.89 %	15	44.11 %
Cardiogenic shock	2	4.2 %	6	17.64 %
Dengue shock	11	23.40 %	6	17.64 %

Figure 7

Distribution of outcome according to etiology



Out of 34 non survivors, Mortality rate is higher among septic shock (44.11 %) followed by hypovolemic and cardiogenic shock.

Table 8 Abnormal Renal function tests -Distribution

Etiology	Abnormal RFT	
	Number	**Percentage**
Hypovolemic shock	7	30.43%
Septic shock	9	39.13 %
Cardiogenic shock	3	13.04%
Dengue shock	4	17.39%
Total cases	23	**100 %**

Table 9 : Distribution Of Severity of Shock

Etiology	Compensated shock	Decompensate shock	Total cases
Hypovolemic shock	21 (56.75%)	13(29.54 %)	34
Septic shock	5 (13.51 %)	17(38.63 %)	22
Cardiogenic shock	1(2.7%)	7(15.90%)	8
Dengue shock	10(27.02%)	7 (15.90%)	17
Total cases	**37(100 %)**	**44 (100%)**	**81**

Out of 81 cases,37 cases (45.67%) presented in compensated stage, while 44 cases(54.32%) presented in decompensated state. out of the decompensated shock, septic shock constitutes the majority (38.63%).Abnormal RFT noted in 28.39% cases excluding cases where RFT not sent.

Table 10 : Outcome distribution according to severity

	COMPENSATED SHOCK	**DECOMPENSATED SHOCK**
SURVIVORS	37 (100 %)	10(22.72%)
NON SURVIVORS	0	34(77.27%)
TOTALCASES	**37**	**44**

Table 11: Comparison of mechanical ventilation among survivors and non survivors

VENTILATIONS	**SURVIVORS**		**NON SURVIVORS**	
	NUMBER	**%**	**NUMBER**	**%**
YES	3	6.3 %	29	85.29%
NO	44	93.61 %	5	14.70%

Table 12: Etiology Distribution

ETIOLOGY	TOTAL CASES
HYPOVOLEMIC SHOCK	**34**
Acute gastroenteritis	**32**
Diabetic ketoacidosis	**1**
Kerosene poisoning	**1**
SEPTIC SHOCK	**22**
Septicemia	**16**
Meningitis	**4**
Empyema	**1**
Gluteal abscess	**1**
CARDIOGENIC SHOCK	**8**
Myocarditis	**4**
Dilated cardiomyopathy	**1**
Rheumatic heart disease	**1**
Scorpion sting	**1**
Chronic renal failure	**1**
DENGUE SHOCK	**17**

Figure - 8

Comparision Of Severity Of Shock

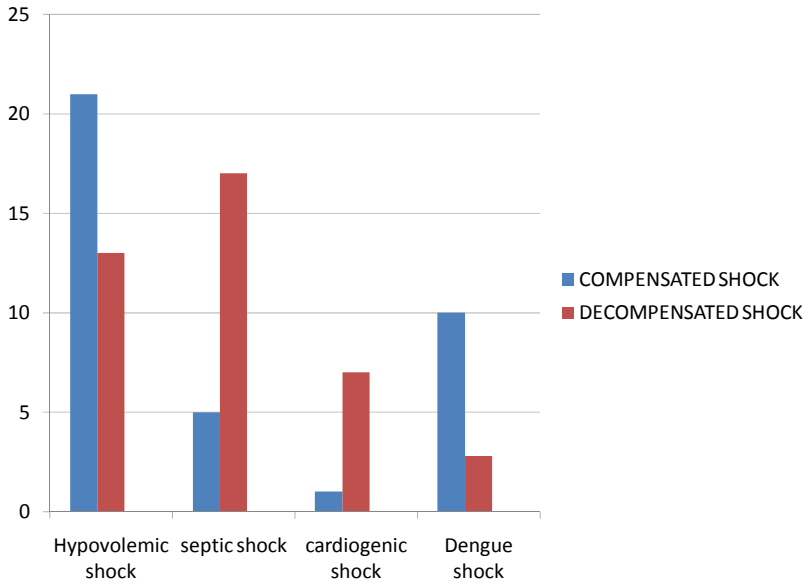
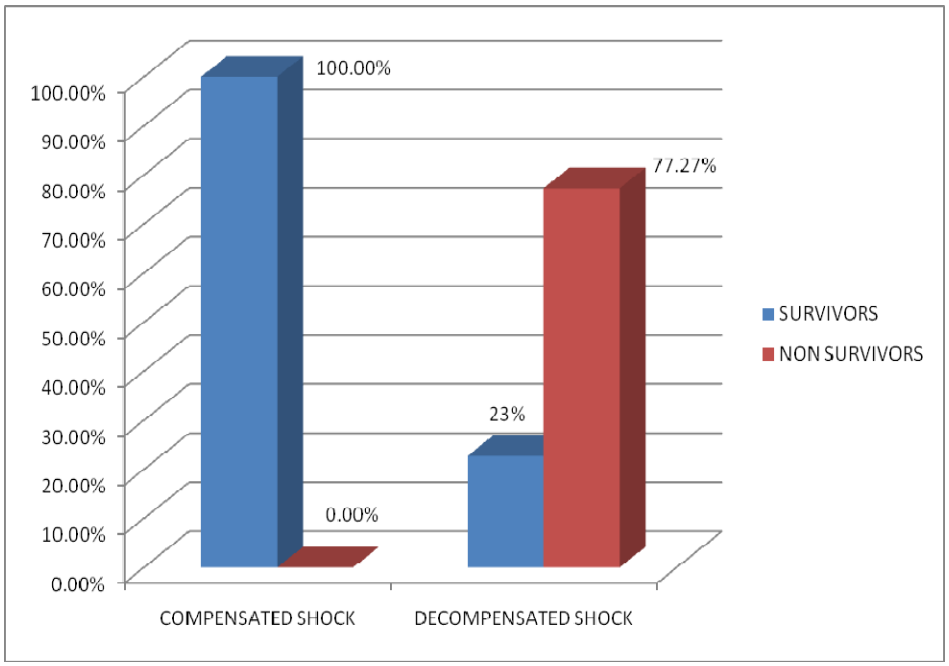


Figure – 9

Outcome according to Severity



DISCUSSION

Shock is one of the most common emergencies in pediatrics. In our study it is accounted for 81 /7095 admissions in Pediatric ward. In a study done by Daljit Singh et al it accounted for 4.5% of PICU admissions.[63]

In our study most common age group was less than one year (accounting for 34.56%) followed by 1-5years (33.33%).

In our study male patients constituted about 49.38% and female patients 50.61%. This is not in accordance to study by Praveen Khilani et al in which males constituted 60%, which was mainly due to male dominated society in India.[64]

In this study the overall mortality in shock was 41.97% (34/81) which is in concordance with that found in the literature (30-60%).[1,39] In a study done by Daljith Singh et al mortality was 26.4%.[63]

There was no significant influence of sex on the out come in present study. Similar findings have been observed in Daljit Singh et al study.[63] But ,children less than 1 year showed higher mortality rate and children 6-10 years showed higher survival rate. De Freitas and Aragao et al found out that in children admitted to ICU mortality was higher in children less than 2 years of age.[65] And in a study by Chang P et al on

non-traumatic shock cases, age less than 2 years tend to have poor outcome.[39] In a study done by Kumar et al, on triage scoring of severity of illness in SIRS children, the mortality increased with decreasing age.[66]

In this study, Hypovolemic shock is the most common cause of shock 34/81(41.97%) followed by septic shock 22/81(27.16%), Dengue shock 17/81(20.98%) and cardiogenic shock 8/81(9.8%).

Hypovolemic shock is the most common cause of shock in children as noted in various other studies ,is also noted in our study.[2-4,10] Similarly in other study by Chang P et al, it accounted for 7/22(32%) of the cases admitted with shock.[39]

The mortality in shock depends on the etiology.[2] In this study septic shock had maximum mortality 44.11 % (15/34) whereas in other studies it ranged from 10-82% in the children,[16,33-39] and 40-60% in adults.[20,38] In a study done by Daljith Singh et al, septic shock has got mortality of 46.7%.[63]

The most common infections of septic shock were Acute CNS infection and pneumonia. Similarly respiratory infection was noted in the other study by Watson et al[16], where as Jacob et al has reported meningitis as the most common cause of septic shock.[68] Of the survived cases, 7

cases of septic shock had positive culture reports.of those seven cases,3 cases grown Coagulase negative staphylococcus aureus,2cases grown Escherichia coli,1 grown Klebsiella species and other grown b-hemolytic streptococci.

Cardiogenic shock was found to have mortality of 75% (6/8). The most common cause of cardiogenic shock in our study were viral myocarditis and congenital heart diseases.. In Daljit Singh et al[63] Congenital heart disease was the most common cause of cardiogenic shock 53%. In a study by Chang P et al mortality was 75% in cardiogenicshock[39] and cardiogenic shock due to myocarditis varied from 2-37% in two studies.[69,70]

The mortality due to acute rheumatic fever was 2% in a study by Majeed HA et al.[71] The mortality of congenital heart disease patients admitted in ICU was 24.6% as reported by Kapil D et al.

Hypovolemic shock had a least mortality in this study 7/34 (20.58%), similar to that found in literature 0-20%.[39,74,75] In a study done by Daljit Singh et al[63] mortality due to hypovolemic shock was 2.3%. Acute gastroenteritis was the most common cause of hypovolemic shock in this study as was found in a study by Chang P et al[39] and also according

to WHO which states acute diarrhoeal disease is one of the most common causes of mortality in children.[19]

Temporal patterns of various clinical parameters showed significant differences in some parameters between survivors and non-survivors. The general trend is towards normalization of various physiological variables in survivors in the first 24-48hours. Where as the variables tended to be abnormal in non-survivors.

GCS at admission was significantly low in non-survivors (7.85±2.73) than in survivors (13.02±1.99), p value is statistically significant (<0.001). Similarly in study done by Raicevic R et al, level of consciousness was in positive correlation with outcome[78], and GCS <8 was an independent predictor of mortality in a new prognostic scoring system for meningococcal shock.[79]

On admission, Heart rate (mean ± SD) – survivors (133.17 ± 23.25), non-survivors (156.23 ± 34.02), p<0.001, which is statistically significant.

There were more ventilated patients in the non-survivors (85.29%) than in survivors (6.3%) (p=0.001). Need for mechanical ventilation predicted mortality in shock cases because of two reasons 1) the need for

mechanical ventilation per se indicated the severity of shock 2) the multiple complications associated with ventilation which contribute to the mortality. The need for mechanical ventilation is found to be independent risk factor for mortality in this study.

Hypoglycemia (blood glucose <50mg%) were noted in 11 cases. Out of them 10 cases survived and only 1 died.

Abnormal renal function test were noted in 23 cases. Out of which septic shock constitutes 39.13% and hypovolemic shock constitute 30.43%.

Compensated shock states were noted in 37 cases. Out of which all 37(100%) survived. Decompensated shock states were noted in 44 cases. Out of which only 10(22.72%) survived.

In the compensated shocks states, a majority were noted in hypovolemic shock (56.75%). The decompensated shocks states, a majority were noted in septic shock (38.63%).

CONCLUSION

1. A total of 81 cases who met the definition of shock among 7095 patients admitted to the ward during the study period which constituted 1.14% admissions.
2. Majority of cases are in the age group of < 1 year (34.16%).
3. There was no significant difference in the sex distribution in the survivors and non-survivors, while children among 6-10 years showed higher survival rate.
4. Out of 81 cases of shock in this study, hypovolemic shock(41.97%) was the most common cause of shock followed septic(27.16%) ,Dengue(20.98%) and cardiogenic shock(9.8%).The most common infection of septic shock were pneumonia and neuroinfection . The most common cause of cardiogenic shock were myocarditis and congenital heart disease and for hypovolemic shock was gastroenteritis.
5. In this study overall mortality of shock was 41.97%. Septic shock has got highest mortality (68.18%) and hypovolemic shock has got least mortality (20.58%).

6. Temporal patterns of various clinical parameters showed a trend towards normalization of the various physiological variables in survivors in the first 24-48 hours where as the variables tend to be abnormal in non-survivors.
7. The clinical variables at admission which were significantly different between survivors and non survivors were:

• GCS (mean±SD) – survivors (13.02±1.99), non-survivors (7.85±2.73), p value <0.001.

• Heart rate (mean±SD) – survivors (133.17±23.25), non-survivors (156.23±34.02), p<0.001.

There was increased need of mechanical ventilator in non-survivors (85.29%) as compared to survivors (6.3%), (p=0.001).

Septic shock has got highest mortality (65.5%) followed by cardiogenic shock (31.0%) and hypovolemic shock has got least mortality (3.4%).

The clinical variables at 24 hrs after admission, which were significantly different between survivors and non-survivors, were heart rate. Mean arterial blood pressure, capillary refilling time, core and peripheral temperature gradient and urine out put.

RECOMMENDATIONS

- Continuous hemodynamic monitoring is essential in all cases of shock. Central venous pressure monitoring were not needed in all cases of shock at resource limited settings.
- Early referral of cases diagnosed to have shock will improve the outcome.
- Hypovolemic shock due to acute gastroenteritis is common. Measures to implement oral rehydration therapy should be intensified at primary health centres and sub centres.
- Early goal directed therapy should be implemented in all cases.

LIMITATIONS

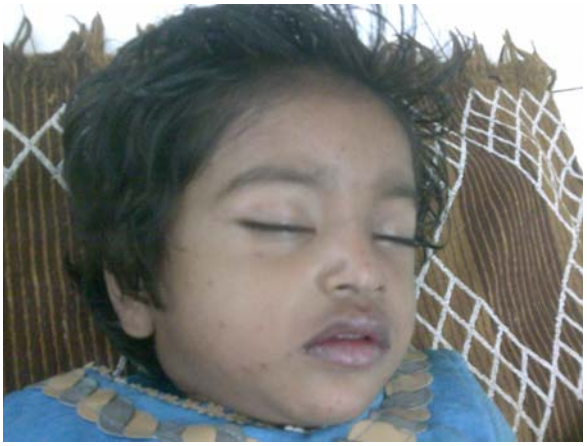
- Markers of shock such as IL1, NO etc., were not done due to limited resources.
- Co-morbid biochemical parameters such as hypoglycemia, dyselectrolemia, hyperlactemia and arterial blood gas analysis influencing mortality due to shock were not assessed.
- Efficacy of bedside ultrasound abdomen, Echo, CVP Monitoring should be evaluated by further studies.

CLINICAL PROFILE OF SHOCK IN CHILDREN AT GRH

NAME	AGE	SEX	I.P.NO	D.O.A	DIAGNOSIS	HR	RR	BP	SPO2 %	GCS	CRT	URINE OUTPUT	INOTROPES	VENTILATION	HB %	TC	DC	SUGAR	RFT	BLOOD CULTURES	OUTCOME	TYPE	SEVERITY
jeyaram	6 months	male	84365	4/10/2009	AGE SHOCK	0hrs 12 24 160 152 138	0hrs 12 24 40 38 38	0hrs 24hrs 90/60 90/60	92	0hrs 24hrs 9/15 15/15	0hrs 24hrs > 3 sec < 3 sec	passed after 6 hrs	NIL	NIL	8 gms	8600	P60 L36 E2 M2	56	N	NOT DONE	SURVIVED	HYPOVOLEMIC SHOCK	COMPENSATED
Karthick kumar	9 years	male	84180	3/10/2009	scorpion sting -shock	130 126 120	32 30 28	90/60 100/70	92	12/15 15/15	>3 sec <3 sec	passed after 3 hrs	NIL	NIL	9.2 gms	10400	P52 L38 E5 M2	42	N	NOTDONE	SURVIVED	CARDIOGENIC SHOCK	DECOMPENSATED
Nagaraj	2 years	male	85603	8/10/2009	AGE SHOCK	142 128 120	42 30 22	80/40 90/60	94	10/15 15/15	>3sec <3sec	passed after 3 hrs	NIL	NIL	6.8	8600	P62 L34 E2 M2	82	ABNORMAL	NOTDONE	SURVIVED	HYPOVOLEMIC SHOCK	COMPENSATED
Afrin banu	46 days	female	86210	11/10/2009	AGE SHOCK	180 - -	irregular	NR	NR	5/15 --------	> 3 SEC	NIL	DOPAMINE 3 hrs	for 3 hrs	-	-	-	-	-	NOTDONE	DIED	HYPOVOLEMIC SHOCK	DECOMPENSATED
yogabharath	6 months	male	87480	16/10/2009	meningitis -shock	176 - -	shallow	NR	NR	6/15 ------	>3 sec	NIL	DOP 5 HRS	FOR 6 HRS	-	-	-	-	-	NOTDONE	DIED	SEPTIC SHOCK	DECOMPENSATED
Thaswin	8 months	male	88618	20/10/2009	acute CNS INF-SHOCK	182 - -	30 - -	60/--	NR	4/15 ----------	>3 sec	NIL	DOP 2 HRS	FOR 3 HRS	-	-	-	-	-	NOTDONE	DIED	SEPTIC SHOCK	DECOMPENSATED
Gayathri	9 years	female	92462	28/10/2009	DKA SHOCK	140 150 -	42 - -	90/40 ----	86	10/15 -----	> 3SEC	NIL	DOP 10 HRS	for 3 hrs	-	-	-	380	ABNORMAL	NOTDONE	DIED	HYPOVOLEMIC SHOCK	DECOMPENSATED
Iswarya	2 months	female	92391	3/11/2009	AGE SHOCK	183 160 120	38 36 36	60 /-- 80/60	82 92	12/15 14/15	>3 SEC < 3SEC	passed after 3 hrs	-	-	9.2	7600	P54 L42 E 4	60	ABNORMAL	NOTDONE	SURVIVED	HYPOVOLEMIC SHOCK	DECOMPENSATED
Rabecca	2months	female	93309	6/11/2009	myocarditis -shock	202 - -	unstable	NR	NR	4/15 -------	>3sec <3sec	NIL	DOP 12 HRS	FOR 10 HRS	-	-	-	66	ABNORMAL	NOTDONE	DIED	CARDIOGENIC SHOCK	DECOMPENSATED
Soundarya	5 years	female	93473	7/11/2009	DENGUE -SHOCK	80 100 96	24 22 20	80/40 90/60	92	13/15 15/15	>3sec <3sec	passed after 3 hrs	-	-	8.4	5400	P36 L52 E2 M1	60	ABNORMAL	NOTDONE	SURVIVED	DENGUE SHOCK	COMPENSATED
Prisilla	8 months	female	93984	9/11/2009	septic shock	170 - -	gasping	NR	NR	4/15 ------	>3 SEC	NIL	DOP 45 MIN	FOR 30MIN	-	-	-	-	-	NOTDONE	DIED	SEPTIC SHOCK	DECOMPENSATED
Deepika	2 1/2 years	female	93985	11/11/2009	DENGUE -SHOCK	108 - -	28 24 -	60/40 -----	90	12/15 -----	>3 SEC	NIL	DOP 10 HRS	-	-	-	-	68	ABNORMAL	NOTDONE	DIED	DENGUE SHOCK	DECOMPENSATED
Gopika	3 years	female	95026	13/11/2009	DENGUE -SHOCK	140 120 118	32 30 26	90/40 90/60	94	13/15 15/15	>3sec <3sec	PASSED AFTER 4 HRS	-	-	10.2	7200	P38 L52 E3M2	80	NORMAL	NOTDONE	SURVIVED	DENGUE SHOCK	COMPENSATED
Karpoora sundar	2 1/2 years	male	97951	24/11/2009	DENGUE -SHOCK	72 82 90	30 - -	NR	NR	9/15 -----	>3 SEC<3SEC	NL	DOP 45 MIN	FOR 45 MIN	-	-	-	-	-	NOTDONE	DIED	DENGUE SHOCK	DECOMPENSATED
Tharunpandi	2 years	male	100020	1/12/2009	AGE SEPSIS SHOCK	138 - -	32 - -	NR	NR	12/15 -----	>3 SEC	NIL	DOP 4 HRS	for 3 hrs	-	-	-	-	-	NOTDONE	DIED	SEPTIC SHOCK	DECOMPENSATED
Meena	5 years	female	101528	5/12/2009	DENGUE -SHOCK	100 108 110	30 28 20	90/40 100/60	90	13/15 15/15	>3sec <3sec	passed after 3 hrs	-	-	7.8	7500	P52 L38 E5 M2	70	N	NOTDONE	SURVIVED	DENGUE SHOCK	DECOMPENSATED
Gayathri	10months	female	102759	9/12/2009	DENGUE -SHOCK	90 110 108	42 38 30	80/60 80/60	92	10/15 12/15	>3sec <3sec	PASSED AFTER 5 HRS	DOP FOR 24 HRS	-	8.2	8600	P40 L58 E1M0	80	N	NOT DONE	SURVIVED	DENGUE SHOCK	DECOMPENSATED
Shanmuga priya	50 days	female	103037	12/12/2009	septic shock	160 152 148	32 30 30	80/60 80/60	94	10/15 14/15	>3sec <3sec	PASSED AFTER 4 HRS	DOP FOR 30 HRS	-	7.2	10800	P62 L34 E2 M2	72	ABNORMAL	KLEBSIELLA SPECIES +	SURVIVED	SEPTIC SHOCK	COMPENSATED
palaniswmy	6 years	male	103289	13/12/2009	DENGUE -SHOCK	120 116 112	28 26 28	100/60 100/60	98	13/15 15/15	>3sec <3sec	PASSEDAFTER 6 HRS	-	-	10.2	10600	P66 L28 E3	60	NORMAL	NOTDONE	SURVIVED	DENGUE SHOCK	COMPENSATED
hariharan	7 YEARS	male	103511	14/12/2009	DENGUE -SHOCK	80 60 100	24 22 24	100/60 110/70	92	13/15 15/15	>3sec <3sec	PASSEDAFTER 3 HRS	-	-	9.6	7200	P38 L60 E3	54	NORMAL	NOTDONE	SURVIVED	DENGUE SHOCK	COMPENSATED
B/O lakshmi	44 days	male	103877	15/12/2009	septic shock	160 - -	42 - -	NR	NR	8/15 -----	>5 SEC ----	NIL	DOP FOR 2 HRS	FOR 3 HRS	-	-	-	-	-	NOTDONE	DIED	SEPTIC SHOCK	DECOMPENSATED
Gowtham	1 3/4 year	male	105125	20/12/2009	septic shock	152 132 128	40 38 36	60/40 70/50	90	9/15 15/15	>3 SEC < 3SEC	PASSED AFTER 3 HRS	DOP FOR 6 HRS	-	9.8	8400	P60 L36 E2 M2	42	N	E.COLI GROWN	SURVIVED	SEPTIC SHOCK	COMPENSATED
Arokiyaraj	2 1/2years m	male	107524	30/12/2009	septic shock	180 - -	42 - -	NR	NR	5/15 -----	>3SEC -----	NIL	DOP FOR 4 HRS	FOR 2 HRS	-	-	-	-	-	NOTDONE	DIED	SEPTIC SHOCK	DECOMPENSATED
meenakshi	5 months	female	599	3/1/2010	age shock seizures	160 - -	gasping	NR	NR	8/15 9/15	>3sec <3sec	NIL	DOP FOR 1 HRS	FOR 1 HRS	-	-	-	-	-	NOTDONE	DIED	HYPOVOLEMIC SHOCK	DECOMPENSATED
Rooban vijay	1 1/4 years	male	1557	6/1/2010	AGE SHOCK	186 - -	48 - -	60/- ----	NR	9/15 -----	>3SEC -----	NIL	DOP FOR 12HRS	FOR2 HRS	-	-	-	-	-	NOTDONE	DIED	HYPOVOLEMIC SHOCK	DECOMPENSATED
Karthiga	12 years	female	2916	12/1/2010	CRF SHOCK	130 140 132	40 38 42	80/60 90/60	NR	10/15 -----	>3SEC------	NIL	DOP FOR 20 HRS	FOR 3 HRS	6.4	7600	P56 L38 E 2	50	ABNORMAL	NOTDONE	DIED	CARDIOGENIC SHOCK	DECOMPENSATED
VELAVAN	3 years	male	3375	14/1/2010	septic shock	142 128 132	42 36 38	60/40 70/50	86	10/15 13/15	>3SEC	PASSED AFTER 8 HRS	DOP FOR 48 HRS	FOR 4 HRS	8.2	5400	P62 L34 E2 M2	62	ABNORMAL	CONS GROWN	DIED	SEPTIC SHOCK	DECOMPENSATED
Muneeshwaran	9 months	male	4152	17/1/2010	septic shock	160 132 140	36 32 34	NR	NR	8/15 9/15	>3SEC -----	NIL	DOP FOR 48 HRS	FOR 8 HRS	9	18,200	P80L20	52	ABNORMAL	NOTDONE	DIED	SEPTIC SHOCK	DECOMPENSATED
nithya	8 years	female	5158	21/1/2010	DENGUE -SHOCK	102 110 108	32 30 32	100/70 100/70	92	14/15 15/15	>3sec <3sec	passed after 3 hrs	-	-	10	8200	P46 L48 E2	50	N	NOTDONE	SURVIVED	DENGUE SHOCK	COMPENSATED
Kaleeshwari	8 years	female	6321	25/1/2010	AGE SHOCK	128 118 120	28 26 24	110/70 110/70	92	15/15 15/15	>3sec <3sec	PASSED AFTER 2 HRS	-	-	8.2	9600	p68 L26 E2	62	N	NOTDONE	SURVIVED	HYPOVOLEMIC SHOCK	COMPENSATED
Shobana	12 years	female	6638	27/1/2010	RHD SHOCK	130 120 122	38 38 32	130/80 120/70	99	15/15 15/15	>3sec <3sec	PASSED AFTER 3 HRS	DOP FOR 5 HRS	FOR 5 HRS	9	10200	P40 L58 E1M0	70	N	NOTDONE	SURVIVED	CARDIOGENIC SHOCK	COMPENSATED
Sivakumar	7 years	male	11200	1/2/2010	AGE SHOCK	130 128 120	42 38 32	110/70 110/70	92	15/15 15/15	>3sec <3sec	PASSED AFTER 3 HRS	-	-	8.6	7200	P46L52 E 2	62	N	NOTDONE	SURVIVED	HYPOVOLEMIC SHOCK	COMPENSATED
Lekhasree	1 1/2 year	female	12600	19/2/2010	kerosene pois shock	160 150 140	42 38 30	NR	NR	10/15 ------	>3SEC -----	NIL	DOP FOR 4 HRS	FOR 4 HRS	9.2	8600	P42 L56 E2	70	ABNORMAL	NOTDONE	DIED	HYPOVOLEMIC SHOCK	DECOMPENSATED
Nagapandi	2 1/2 years	male	19104	18/3/2010	septic shock	162 - -	46 - -	60 /-	90	13/15 ----	>3SEC -----	NIL	DOPFOR 5 HRS	FOR 2HRS	8.6	8200	P60 L36 E2 M2	82	ABNORMAL	NOTDONE	DIED	SEPTIC SHOCK	DECOMPENSATED
Tharunya	6 years	female	19664	20/3/2010	empyema shock	140 - -	42 - -	100/60 -----	96	15/15	>3SEC <3SEC	passed after 3 hrs	DOP FOR 24 HRS	-	9	10800	P72 L28	56	ABNORMAL	NOTDONE	SURVIVED	SEPTIC SHOCK	COMPENSATED
Shobika	33 days	female	22568	30/3/2010	septic shock	138 - -	30 - -	NR	NR	10/15 ----	>4SEC ----	NIL	DOPFOR 5 HRS	FOR 2 HRS	-	-	-	-	-	NOTDONE	DIED	SEPTIC SHOCK	DECOMPENSATED
B/O Palaniselvi	2 1/2 month	male	23298	2/4/2010	septic shock	160 - -	32 - -	NR	NR	3/15 ----	>4 SEC ---	NIL	DOP FOR1 HR	FOR 1HR	-	-	-	-	-	NOTDONE	DIED	SEPTIC SHOCK	DECOMPENSATED
Ritsen	1 year	male	24621	7/4/2010	AGE SHOCK	168 156 140	38 32 34	90/60 90/60	96	15/15	>3SEC <3SEC	passed after 3 hrs	-	-	9.2	9200	p48 L46 E2	42	N	NOTDONE	SURVIVED	HYPOVOLEMIC SHOCK	COMPENSATED
Arunadevi	9 years	female	25349	10/4/2010	DENGUE -SHOCK	142 140 130	40 30 28	100/60 110/70	96 96	15/15 15/15	>3sec <3sec	PASSED AFTER 2 HRS	-	-	10.4	10200	P42 L56 E2	60	N	NOTDONE	SURVIVED	DENGUE SHOCK	COMPENSATED
Aravindh	6 years	male	27074	16/4/2010	AGE SHOCK	132 134 130	30 26 20	100/60 100/60	96 96	15/15 15/15	>3sec <3sec	PASSED AFTER 3 HRS	-	-	6.8	9420	P51 L36 E3	80	N	NOTDONE	SURVIVED	HYPOVOLEMIC SHOCK	COMPENSATED
Rajkumar	7 years	male	28297	21/4/2010	AGE SHOCK	128 120 116	30 32 30	100/60 100/70	98 98	15/15 15/15	>3sec <3sec	PASSEDAFTER 2 HRS	-	-	9.8	11200	P80 L18 E2	112	N	NOTDONE	SURVIVED	HYPOVOLEMIC SHOCK	COMPENSATED
Sanchana	7 mon	female	29363	25/4/2010	septic shock	164 - -	32 - -	NR	NR	6/15 ----	>3SEC	NIL	DOP FOR 6 HRS	-	-	-	-	-	-	NOT DONE	DIED	SEPTIC SHOCK	DECOMPENSATED
Kani	8 years	male	32294	2/5/2010	AGE SHOCK	132 130 130	28 24 20	90/50 100/60	92 98	13/15 15/15	>3sec <3sec	PASSED AFTER5 HRS	-	-	10.6	8600	P28 L72 E2	116	N	NOTDONE	SURVIVED	HYPOVOLEMIC SHOCK	DECOMPENSATED
Mahalaxmi	4 years	female	36394	9/5/2010	AGE SHOCK	140 128 110	30 28 20	90/60 100/60	96 98	12/15 15/15	>3SEC <3SEC	PASSEDAFTER 4 HRS	-	-	8.2	12400	P32L64E2M2	24	N	NOTDONE	SURVIVED	HYPOVOLEMIC SHOCK	DECOMPENSATED
Vetrivel	7 months	male	38313	12/5/2010	AGE SHOCK	162 - -	40 - -	NR	NR	5/15 --------	>4 SEC ---	NIL	DOP FOR 10 HRS	FOR 6 HRS	-	-	-	-	-	NOTDONE	DIED	HYPOVOLEMIC SHOCK	DECOMPENSATED
Devipriya	1 1/2 year f	female	38782	24/5/2010	AGE SHOCK	124 120 118	30 24 20	100/60 100/60	92 96	15/15 15/15	>3sec <3sec	PASSEDAFTER4 HRS	-	-	10.2	12800	P40 L58 E1M0	80	N	NOTDONE	SURVIVED	HYPOVOLEMIC SHOCK	COMPENSATED
PRIYADHARSINI	8 years	female	41167	2/6/2010	AGE SHOCK	134 120 108	33 27 28	100/60 100/60	98 98	14/15 15.15	>3sec <3sec	PASSEDAFTER3 HRS	-	-	6.8	10200	P28 L72 E2	60	N	NOTDONE	SURVIVED	HYPOVOLEMIC SHOCK	COMPENSATED
Velmani	5 years	male	41924	4/6/2010	meningitis shock	140 132 120	42 36 30	90/60 100/70	92 94	13/15 15/15	>3sec <3sec	PASSEDAFTER1 HRS	-	FOR 6 HRS	9.2	8600	P62 L34 E2 M2	62	ABNORMAL	CONS GROWN	SURVIVED	SEPTIC SHOCK	COMPENSATED
Muthuselvam	1 1/2 year	male	43047	8/6/2010	AGE SHOCK	128 126 120	40 32 28	90/60 90/60	90 92	14/15 15/15	>3sec <3sec	PASSEDAFTER 3 HRS	-	-	8.6	6800	P72L34E2M2	48	N	NOTDONE	SURVIVED	HYPOVOLEMIC SHOCK	COMPENSATED
B/O Varghese	40 days	female	46283	20/6/2010	AGE SHOCK	160 152 148	32 30 28	80/60 80/60	94 96	13/15 15/15	>3sec <3sec	PASSED AFTER2 HRS	-	-	9	7800	p68 L26 E2	50	N	NOTDONE	SURVIVED	HYPOVOLEMIC SHOCK	COMPENSATED
Kavyashree	4 months	female	46878	22/6/2010	myocarditis -shock	208 - -	48 - -	NR	NR	5/15 -----	>4SEC ----	NIL	DOPFOR 3HRS	FOR 2 HRS	-	-	-	-	-	NOTDONE	DIED	CARDIOGENIC SHOCK	DECOMPENSATED
Mahalaxmi	40 days	female	47513	25/6/2010	AGE SHOCK	168 154 140	42 38 30	80/60 80/60	92 94	13/15	>3sec <3sec	PASSED AFTER 1HR	-	-	10.2	5600	p68 L26 E2	42	N	NOTDONE	SURVIVED	HYPOVOLEMIC SHOCK	COMPENSATED
Nagaraj	4 years	male	48236	27/6/2010	DENGUE -SHOCK	142 - -	32 - -	90/60 90/60	86	12/15 ---	>3SEC -----	NIL	DOP FOR 1 HRS	FOR 1 HRS	-	-	-	-	-	NOTDONE	DIED	DENGUE SHOCK	DECOMPENSATED
KISHORE	2 years	male	48803	29/6/2010	AGE SHOCK	150 142 138	38 35 30	90/60 90/60	92 94	14/15 15/15	>3SEC <3SEC	PASSED AFTER 4 HRS	-	-	8.2	6800	P78L30E2	46	N	NOTDONE	SURVIVED	HYPOVOLEMIC SHOCK	COMPENSATED
Nithyarupa	6 years	female	48859	29/6/2010	AGE SHOCK	138 136 130	32 28 22	100/70 100/70	94 96	14/15 15/15	>3sec <3sec	PASSEDAFTER 2 HRS	-	-	9.6	5600	P68L32 E2	62	N	NOTDONE	SURVIVED	HYPOVOLEMIC SHOCK	COMPENSATED
Anand	11 years	male	50090	4/7/2010	AGE SHOCK	120 118 116	26 22 20	100/70 100/70	92 96	14/15 15/15	>3sec <3sec	PASSEDAFTER 4 HRS	-	-	10.2	8400	P72 L36 E2	58	ABNORMAL	NOTDONE	SURVIVED	HYPOVOLEMIC SHOCK	COMPENSATED
B/O Anish	30days	female	50136	4/7/2010	septic shock	160 140 138	32 30 28	NR	NR	5/15 ---	>4SEC ----	NIL	DOP FOR 48 HRS	FOR 4 HRS	6.2	10200	P86L12 E2	40	ABNORMAL	E.COLI GROWN	DIED	SEPTIC SHOCK	DECOMPENSATED

Karupasamy	2 1/2years m	male	50142	4/7/2010	AGE SHOCK	152 150 130	38 30 22	90/60 90/60	92 96	13/15 15/15	>3SEC <3SEC	PASSEDAFTER 2 HRS	-	-	8.6	7600	P78L26E2 M2	60	N	NOTDONE	SURVIVED	HYPOVOLEMIC SHOCK	COMPENSATED
Narmatha	8 years	female	51024	7/7/2010	AGE SHOCK	118 110 102	30 24 20	100/70 100/70	92 94	14/15 15/15	> 3 sec < 3 sec	PASSEDAFTER 3 HRS	-	-	9.2	10800	P64L32 E2	62	N	NOTDONE	SURVIVED	HYPOVOLEMIC SHOCK	COMPENSATED
Kaviyarasan	7 months	male	104146	16/12/2009	DENGUE -SHOCK	88 100 102	18 16 16	90/60 90/60	96 96	15/15 15/15	>3sec <3sec	PASSED AFTER 2 HRS	-	-	10	8200	P52 L46 E2	62	N	NOTDONE	SURVIVED	DENGUE SHOCK	COMPENSATED
Karuppuraja	10 year	male	53859	17/7/2010	AGE SHOCK	122 120 110	22 20 18	100/70 100/70	92 96	15/15 15/15	>3sec <3sec	PASSED AFTER 4 HRS	-	-	8.2	6400	P72 L26 E2	54	N	NOTDONE	SURVIVED	HYPOVOLEMIC SHOCK	COMPENSATED
Selvakumar	4 months	male	53950	18/7/2010	AGE SHOCK	148 128 110	30 24 22	90/60 90/60	96 98	14/15 15/15	>3sec <3sec	PASSEDAFTER 5 HRS	-	-	9.6	10200	P64L32 E2	50	N	NOTDONE	SURVIVED	HYPOVOLEMIC SHOCK	COMPENSATED
Deepak	10 months	male	53932	18/7/2010	septic shock	160 132 112	28 20 20	90/60 90/60	92 93	13/15 14/15	>3sec <3sec	PASSED AFTER 2 HRS	-	-	8.2	9200	P58L40 E2	62	N	NOTDONE	SURVIVED	SEPTIC SHOCK	COMPENSATED
Somasundaram	10 years	male	54974	22/7/2010	AGE SHOCK	122 120 116	24 16 18	100/70 100/70	94 94	15/15 15/15	>3sec <3sec	PASSED AFTER 3 HRS	-	-	9.2	8600	P60 L36 E2 M2	68	N	NOTDONE	SURVIVED	HYPOVOLEMIC SHOCK	COMPENSATED
Rajasekar	2 years	male	57478	29/7/2010	septic shock	142 -- --	42 -- --	NR	NR	6/15 -------	>4SEC ----	NIL	DOP FOR 4 HRS	FOR 4 HRS	-	-	-	-	-	NOTDONE	DIED	SEPTIC SHOCK	DECOMPENSATED
Oviya	10 years	female	58191	13/8/2010	DENGUE -SHOCK	130 126 128	40 42 38	100/60 100/60	92 96	14/15 15/15	>3sec <3sec	PASSEDAFTER5 HRS	-	-	10.2	7600	P70L28E2	80	N	NOTDONE	SURVIVED	DENGUE SHOCK	COMPENSATED
Sumathi	12 years	female	60078	8/8/2010	AGE SHOCK	128 124 120	42 36 30	100/70 100/70	94 96	15/15 15/15	>3sec <3sec	PASSEDAFTER2HRS	-	-	9.6	8200	P42 L56 E2	62	N	NOTDONE	SURVIVED	HYPOVOLEMIC SHOCK	DECOMPENSATED
Yalini	8 years	female	65584	29/8/2010	DENGUE -SHOCK	90 -- ---	40 -- --	NR	NR	8/15 ----	>4SEC ----	NIL	-	-	-	-	-	-	-	NOTDONE	DIED	DENGUE SHOCK	DECOMPENSATED
Lathigasri	3 years	female	65610	29/8/2010	DENGUE -SHOCK	94 108 92	40 38 48	6O/- 60/-	86 88	10/15 8/15	>3SEC >3SEC	PASSED AFTER4 HRS	DOP FOR 24 HRS	FOR 20 HRS	10.2	7800	p48 L46 E2	72	ABNORMAL	NOTDONE	DIED	DENGUE SHOCK	DECOMPENSATED
Gokulnath	36 DAYS	male	66582	1/9/2010	septic shock	180 --- --	58 -- --	NR NR	NR	8/15 ---	>3SEC------	NIL	DOP FOR 10 HRS	FOR 2 HRS	-	-	-	-	-	NOTDONE	DIED	SEPTIC SHOCK	DECOMPENSATED
Durgadevi	5 years	female	66974	3/9/2010	AGE SHOCK	120 110 108	36 29 24	100/60 100/60	92 98	13/15 15/15	.3SEC <3SEC	PASSED AFTER 3 HRS	-	-	8.8	10600	P63 L36 E2	83	N	NOTDONE	SURVIVED	HYPOVOLEMIC SHOCK	COMPENSATED
Maheshwari	7 months	female	68073	8/9/2010	AGE SHOCK SEIZURES	140 --- ---	48 --- --	NR	NR	5/15 -----	>3SEC------	NIL	DOP FOR 3 HRS	-	-	-	-	-	-	NOTDONE	DIED	HYPOVOLEMIC SHOCK	DECOMPENSATED
Saravanan	5months	male	68417	10/9/2010	myocarditis -shock	206 ---- ---	58 --- --	60/- ----	84 ---	10/15 ----	>4SEC ----	PASSEDAFTER 4 HRS	DOP FOR 4 HRS	FOR 4 HRS	-	-	-	-	ABNORMAL	NOTDONE	DIED	CARDIOGENIC SHOCK	DECOMPENSATED
Ranjithkumar	6 years	male	69187	14/9/2010	DENGUE -SHOCK	120 118 102	38 32 28	100/60 100/60	92 94	14/15 15/15	>3sec <3sec	PASSED AFTER3 HRS	-	-	7.2	7300	P62 L34 E2 M2	47	N	NOTDONE	SURVIVED	DENGUE SHOCK	COMPENSATED
Abhinesh	9 months	male	70228	18/9/2010	AGE SHOCK	140 132 116	56 42 30	70/- 90 /60	84 97	10/15 15/15	>3sec <3sec	PASEEDAFTER2HRS	-	-	9.4	9400	P38 L60 E3	63	ABNORMAL	NOTDONE	SURVIVED	HYPOVOLEMIC SHOCK	DECOMPENSATED
Pandi	2 1/2 years	male	70511	19/9/2010	septic shock	180 170 150	62 56 52	NR 90/60	NR 92	8/15 14/15	>3SEC<3sec	passed after 3 hrs	DOP FOR 24 HRS	-	8.2	8000	P64L32 E2	60	ABNORMAL	b hemolytic streptococci grown	SURVIVED	SEPTIC SHOCK	DECOMPENSATED
Divya	8 years	female	70767	20/9/2010	AGE SHOCK	120 112 102	40 34 26	90/60 100/60	86 96	10/15 15/15	>3sec <3sec	passed after 4 hrs	-	-	9.6	9600	p64l36	63	ABNORMAL	NOTDONE	SURVIVED	HYPOVOLEMIC SHOCK	DECOMPENSATED
Yogasri	23/4 year	female	71188	22/9/2010	cardiomyopathy shock	200 180 ---	50 52 --	NR	NR	10/15 15/15	>3sec <3sec	NIL	DOPFOR 6 HRS	FOR 4 HRS	-	-	-	-	-	NOTDONE	DIED	CARDIOGENIC SHOCK	DECOMPENSATED
Riswan banu	10 months	female	72024	26/9/2010	myocarditis -shock	208 --- ---	60 -- --	60/- -----	84	9/15 ----	>3 SEC ---	NIL	DOP FOR6HRS	FOR 4 HRS	-	-	-	-	-	NOTDONE	DIED	CARDIOGENIC SHOCK	DECOMPENSATED
Hasini	11/4year	female	72105	28/9/2010	acute CNS INF-SHOCK	140 130 118	42 38 28	60/- 90/60	84 94	9/15 14/15	>3SEC <3SEC	PASSED AFTER 5 HRS	DOP FOR 12HRS	FOR 2 HRS	9.2	10450	p48 L46 E2	84	ABNORMAL	CONS GROWN	SURVIVED	SEPTIC SHOCK	DECOMPENSATED
Papammal	11 years	female	72982	30/9/2010	ALL DENGUE SOCK	110 --- ---	32 --- --	NR	NR	8/15 ---	>3SEC -----	NIL	DOP FOR 4 HRS	FOR2 HRS	-	-	-	80	ABNORMAL	NOT DONE	DIED	DENGUE SHOCK	DECOMPENSATED

DENGUE SHOCK SYNDROME



SCORPION STING – CARDIOGENIC SHOCK

PEM - SEPTIC SHOCK