“PROSPECTIVE STUDY OF BLOOD LACTATE LEVELS AS A PROGNOSTIC INDICATOR IN PATIENTS WITH SHOCK ADMITTED TO PICU IN TERTIARY CARE CENTRE”

By

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SDUAHER

DISSERTATION SUBMITTED TO SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION AND RESEARCH, KOLAR, KARNATAKA

In partial fulfilment of the requirements for the degree of

DOCTOR OF MEDICINE
IN
PAEDIATRICS

Under the Guidance of

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to be submitted to the

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ACKNOWLEDGEMENT

Academic excellence can be achieved only with guidance of our teachers. I would like to take this opportunity to express my sincere thanks to all who have helped in bringing this manuscript into reality

With humble gratitude and great respect, I would like to thank my teacher, mentor and guide, ***Dr. KRISHNAPPA J,*** *Professor, Department of Paediatrics, Sri Devaraj Urs Medical College, Kolar, for his able guidance, constant encouragement, immense help and valuable advices which went a long way in moulding and enabling me to complete this work successfully. His humble nature, determination , commitment and hunger for knowledge has been my constant source of inspiration. His affection towards students, ever readiness to teach , the painstaking effort to correct errors and his ocean of knowledge has proven the proverb right " A teacher is better than two books"Without his initiative and constant encouragement this study would not have been possible. His vast experience, knowledge, able supervision and valuable advices have served as a constant source of inspiration during the entire course of my study.*

I would like to express my deep sense of gratitude and humble thanks ***Dr KNV PRASAD Dr. SUDHA REDDY VR, Dr BEERE GOWDA Y C, Dr BHANUCHAND P, Dr BHARATH, Dr MURALI, Dr SHIVARAJA, Dr.NARENDRA ,Dr SRIKANTH AND Dr MANASA SHETTY*** *for their valuable inputs, constant advice and guidance throughout the study.*

*I would like to express my sincere thanks to **Dr. SHASHIDHAR K N,** my co guide Professor, Department of Biochemistry, Sri Devaraj Urs Medical College for his valuable support, guidance and encouragement throughout the study.*

I am very thankful to Dr Chethan for helping me in statistical analysis and for his valuable suggestions

*I would also like to thank my colleagues and friends **Dr Mohammed Yasar and Dr Naveen Kumar,** and all my fellow postgraduates for helping me throughout this journey*

I would like to thank the Almighty and my patients for providing me an opportunity to carry out my study .

I would like to thank my most dearest father Dr Satyanarayana Raju G T who has always been my pillar of strength , my grandmother Smt Honnamma my sister Supriya and all my dearest friends for their constant guidance, love, moral support and blessing..

Dr. SRI RAKSHA S

LIST OF ABBREVIATIONS

ADP	Adenosine Di Phosphate
AMP	Adenosine Mono phosphate
ATP	Adenosine Tri Phosphate
AUC	Area Under The Receiver Operating Characteristic curve
BUN	Blood Urea Nitrogen
CI	Confidence Interval
CI	Cardiac Index
CO	Cardiac Output
CPR	Cardio Pulmonary Resuscitation
CRP	C- Reactive Protein
EGDT	Early Goal Directed Therapy
IO	Intra Osseous
IV	Intra Venous
LDH	Lactate Dehydrogenase
NPV	Negative Predictive Values
OP	Organophosphorus Poison
PICU	Pediatrics Intensive Care Unit
PIM	Pediatric Index of Mortality
PPV	Positive Predictive Values
PPV	Positive Pressure Ventilation
PRISM III Scores	Pediatrics Risk Of Mortality III score
PSI	Physiologic Stability Index
RAAS	Rennin Angiotensin Aldosterone System
ROC	Receiver Operating Characteristic Curve
ScvO2	Central Venous Oxygen Saturation
SIRS	Systemic Inflammatory Response Syndrome
SVR	Systemic Vascular Resistance

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ABSTRACT

BACKGROUND: Shock is one of the most frequent life-threatening conditions which is encountered in pediatrics ICU. The appropriate management of shock in pediatrics includes early recognition of tissue hypoxia and its timely intervention thus preventing shift to anerobic metabolism, metabolic acidosis and cell death.

Lactic acid is a metabolite generated as a result of anaerobic glycolysis. This anaerobic glycolysis occurs when there is hypoxia due to inadequate tissue perfusion as occurs in shock. Hyperlactatemia is a very important cardinal finding in pediatric shock. This lactic acid thus estimated can be used as a marker for predicting the outcome of patients in shock. Pediatric risk of mortality III score is a scoring system which is based on the physiologic variables to assess the mortality risk in pediatric ICU patients. Monitoring of patients admitted to pediatric intensive care unit on the basis of PRISM III score and lactate levels is beneficial for the overall outcome and should be incorporated into early resuscitation strategies. As early recognition of shock and aggressive intervention has a better outcome. Our study is intended to know the relation between serum lactate levels and the outcome of the patient and to correlate the same with the PRISM III scores.

OBJECTIVES:

1) **To find out the effectiveness of blood lactate levels as a prognostic indicator of mortality in patients with shock admitted to PICU .**
2) **To correlate between blood lactate levels and PRISM III scores.**

MATERIALS AND METHODS:A prospective observational study was conducted between January 2018 to May 2019, including all the children between the age group of 1 month and 18 years. The children presenting with shock due to any etiology was included in the study. Following a detailed clinical examination and assessing the vital parameters, venous and arterial samples were drawn for relevant investigations including the serum lactate levels.

The serum lactate values were assessed at 0 hours, 12 hours and 24 hours of admission to PICU.

RESULTS: A total of 144 children were included in our study. Majority of the children who presented in shock were below the age group of 1 year. Severe sepsis in septic shock was the most common etiology of shock in children. Bronchopneumonia was the most common cause of septic shock in children. Important biochemical parameters (arterial pH, Bicarbonate, Serum Potassium, BUN and Serum Creatinine) were studied between survivors and non survivors. It was observed that all of these biochemical parameters had a statistical significant difference (p value < 0.001) between survivors and non survivors. Hence all these parameters can be used as prognostic indicators and predictors of mortality in children presenting with shock. Serum lactate levels were assessed between survivors and non survivors at the time of admission 0 hours, 12 hours and 24 hours.There was statistical significant difference in survivors and non survivors at various time intervals (p value <0.001) It was observed that there was persistent hyperlactemia in non survivors and serum lactate values persistently greater than 4mmol/l within the first 24 hours of admission were associated with greater chances of mortality. The sum of the PRISM III scores were compared in between survivors and non survivors.It was observed that the non survivors had persistently elevated PRISM III scores when compared to non survivors.There was statistical significant difference between survivors and non surviors for PRISM III scores at various time intervals (p value < 0.001). Survivors not only had low PRISM III scores initially but later showed a reducing trend. AUC for both serum lactate and PRISM III scores was compared. The area under the ROC curve for the serum lactate levels (0.958) suggests that it was a strong predictor of mortality in study subjects when compared to PRISM III score

which had area under the ROC curve 0.866. Area under the ROC curve for Both PRISM III score and serum lactate levels had a significant P value <0.001

CONCLUSION: Serum lactate value is a single independent useful parameter that can be used as a predictor of mortality which is better and more feasible than PRISM III scores which uses many variables and multiple physiological, biochemical and hematological parameters.

KEY WORDS: Shock, Serum Lactate values, PRISM III scores.

INTRODUCTION

INTRODUCTION

Shock is one of the most frequent life-threatening conditions which is encountered in paediatrics ICU. Shock is an acute process characterized by the body's inability to deliver adequate oxygen to meet the metabolic demands of vital organs and tissues[1,2]. Hypoxia at the tissue level is unable to support normal aerobic cellular metabolism and shift to the less efficient anaerobic metabolism. When there is an imbalance between oxygen delivery to the tissue and oxygen requirement there is oxygen debt which leads to progressive clinical deterioration and lactic acidosis[3,4].

The appropriate management of shock in pediatrics includes early recognition of tissue hypoxia and its timely intervention thus preventing shift to anerobic metabolism, metabolic acidosis and cell death[2]. Early indicators of mortality in pediatrics patients with shock can be employed to assess and determine the risk of mortality so that early intervention can be followed in order to prevent adverse events[5].

Paediatric risk of mortality III score is a scoring system which is based on the physiologic variables to assess the mortality risk in paediatric ICU patients. It consists of 17 physiological variables and 26 sub categories. An increase in the value of the score indicates poor prognosis[2,5].

Lactic acid is a metabolite generated as a result of anaerobic glycolysis. This anaerobic glycolysis occurs when there is hypoxia due to inadequate tissue perfusion as occurs in shock. Hyperlactatemia is a very important cardinal finding in pediatric shock[1,2]. The mechanism of hyperlactemia has two pathways, one in sepsis and the other one in cases septic shock. In case of sepsis an increase in lactate levels implies increased glycolysis due to increase in metabolic rate and in cases of shock the raised glycolytic flux is due to tissue hypoxia. Thus, this implies that there are two varieties of lactate, that is “stress lactate” and “shock lactate”[5,6].

This lactic acid thus estimated can be used as a marker for predicting the outcome of patients in shock[7].

In children admitted with shock when the tissue oxygen requirement is not met by the oxygen delivered to the tissue there will be compensatory increases in the oxygen extraction. If this imbalance between oxygen requirement and oxygen delivery continues there is progressive hypoxia at the tissue level and this leads to compensatory oxygen debt[8,9].

In shock the failure to supply oxygen to meet the demand causes tissue hypoxia and, increased anaerobic metabolism and lactate production.

The tissue hypoxia and anaerobic metabolism are important markers of inadequate tissue perfusion in patients who are in shock and result in inadequate lactate clearance leading to multi organ dysfunction syndrome.[8]

Early recognition of children who are at a greater risk for mortality can help in appropriate timely intervention or any significant changes in the management thus helping in the over all outcome. The critical care in paediatrics has changed significantly over the past several decades and there has been no reliable and consistent marker to predict the outcome of children who present in shock. Shock is one of the most important and common condition with which children present in the

Pediatric Intensive care unit[10]. Serum lactate can be considered as an early predictor of mortality in shock so that there is still room for timely intervention.

Monitoring of patients admitted to pediatric intensive care unit on the basis of PRISM III score and lactate levels is beneficial for the overall outcome and should be incorporated into early resuscitation strategies. As early recognition of shock and aggressive intervention has a better outcome. Our study is intended to know relation between serum lactate levels and the outcome of the patient and to correlate the same with the PRISM III scores.

AIMS & OBJECTIVES

OBJECTIVES OF THE STUDY:

1) To find out the effectiveness of blood lactate levels as a prognostic indicator of mortality in patients with shock admitted to PICU .
2) To correlate between blood lactate levels and PRISM III scores.

REVIEW OF LITERATURE

REVIEW OF LITERATURE

SHOCK:

Definition: It is an acute syndrome of cardiovascular dysfunction in which there is failure of the circulatory system to provide adequate oxygen and nutrients to meet the metabolic demands of the vital organs[10].

It is the common clinical pathway for potentially lethal clinical events which includes, extensive trauma, severe hemorrhage and microbial sepsis. There is systemic hypotension either due to reduced cardiac output or due to reduced effective circulating blood volume[8,9] .

The consequences include reduced effective circulating blood volume and tissue hypoxia. Tissue hypoxia leads to inability to support normal aerobic cellular metabolism, which inturn results in a shift to the less efficient anerobic metabolism. As the shock progresses there is acute imbalance between tissue oxygen requirement and oxygen delivery, this inturn leads to inability to compensate for this deficiency in oxygen delivery leading to **progressive clinical deterioration and lactic acidosis**[8,9].

Compensation for hypoxia at tissue level involves a complex set of responses that try to preserve oxygenation of vital organs. The brain is especially sensitive to hypoxia due to its lack of ability for anerobic metabolism[8].

Irrespective of its cause, shock will have a specific pattern of response, pathophysiology along with clinical features and treatments will vary based on the etiology of shock.

Shock occurs in about 2% of all the hospitalized infants, children and adults and the mortality rate varies depending on the etiology and clinical circumstances [10].

It has been observed that most common reason for mortality is not due to the acute hypotensive phase of shock but as a result of complications and multi organ dysfunction syndrome[9,10].

Educational efforts and utilization of standardized management protocols that emphasize on early recognition and intervention with rapid transfer of critically ill children to a Pediatric intensive care unit have led to reduction in the mortality rate for shock[10].

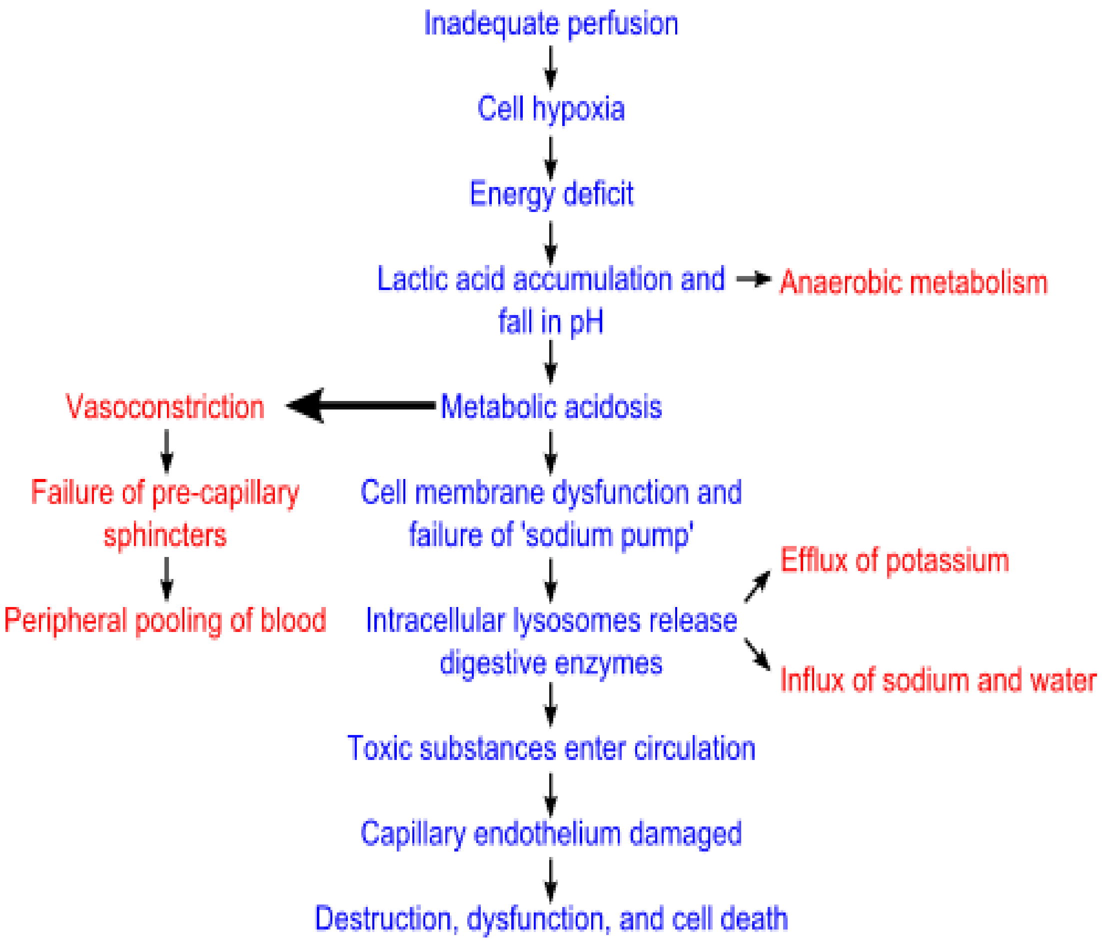


Figure 1: Pathophysiology of Shock[10]

CLASSIFICATION OF SHOCK:

1. **Hypovolemic shock**
2. **Cardiogenic Shock**
3. **Obstructive shock**
4. **Distributive shock**
5. **Septic Shock**

PATHOPHYSIOLOGY OF SHOCK:

Hypovolemic Shock:

It is the most common etiology of shock in children[11]. Volume depletion due to vomiting and diarrhea secondary to gastrointestinal infections are most common etiology. Other causes of hypovolemic shock are hemorrhage (trauma, post-surgical, gastrointestinal), plasma losses(burns, hypoproteinemia, pancreatitis), extra gastrointestinal water losses(glycosuric diuresis, heat stroke) and interstitial third spacing(burns, sepsis ,nephrotic syndrome, protein losing enteropathy and intestinal obstruction)[12]. Acute hypovolemia results in reduced cardiac output due to fall in preload with a compensatory raise in heart rate and systemic vascular resistance. Reduction in the blood pressure which is detected in the baroreceptors of the carotid sinus inturn causes increase in the sympathetic nervous system activity, stimulating cardiac muscle for chronotropic effect and vascular smooth muscle constriction along with epinephrine release from the adrenal medulla. Up regulation of RAAS and release of Anti diuretic hormone from the posterior pituitary gland promote sodium and water retention by the kidneys[11].

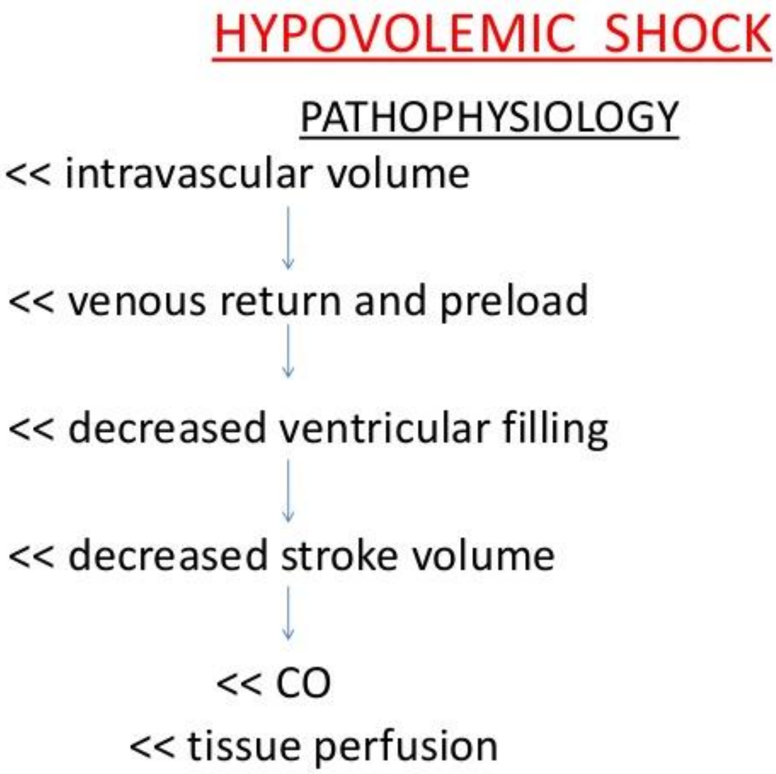


Figure 2: Pathophysiology of hypovolemic shock[10]

Cardiogenic Shock:

The term cardiogenic shock is reserved for decrease in cardiac output due a decrease in myocardial contractility. Shock caused by obstruction of blood flow due to certain types of congenital heart disease is better classified as obstructive shock. Although myocardial depression occurs in all types of shock, primary deficits in myocardial contractility leading on to cardiogenic shock are caused by viral myocarditis, incessant arrythmias, anamolous left coronary artery arising from pulmonary artery, metabolic derangements and postoperative complications of cardiac surgery[11].

Characteristic signs of cardiogenic shock include congestive cardiac failure, including basal crepitations, a gallop rhythm, hepatomegaly, pitting peripheral edema, jugular venous distension, and cardiomegaly on chest X ray[12].

Laboratory findings of elevated troponin, creatinine kinase, or brain natriuretic peptide levels will indicate myocardial dysfunction but they are not universally present. Similar to hypovolemic shock the upregulation of the sympathetic nervous system, RAAS and the natriuretic peptides raise the SVR to compensate for the low cardiac output [12,13].

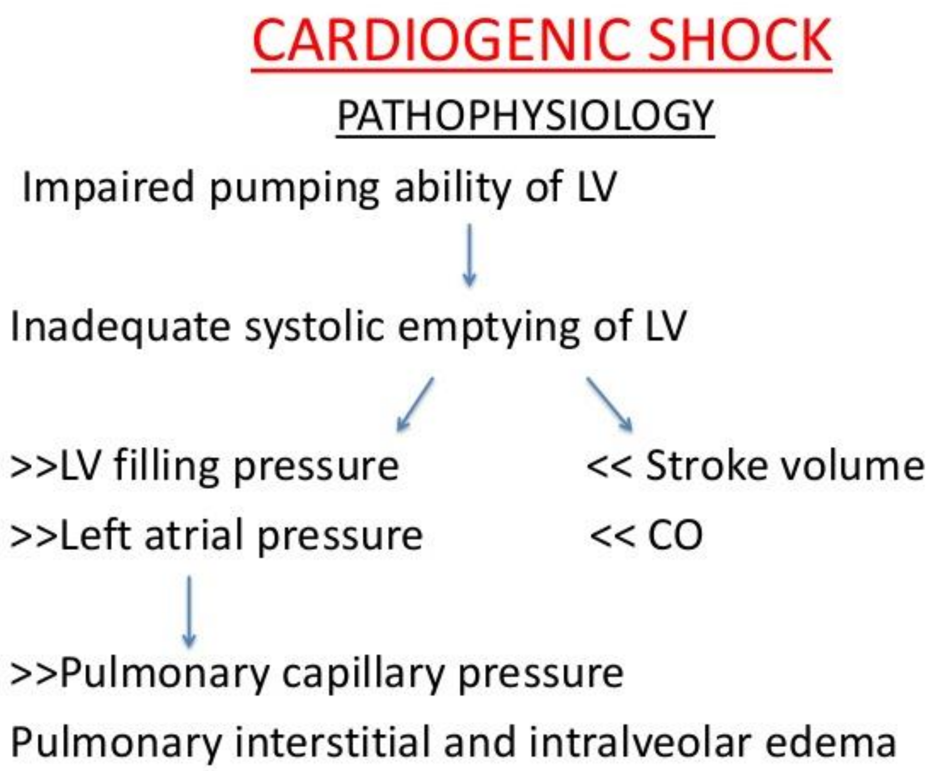


Figure 3: Pathophysiology of Cardiogenic shock[10]

Obstructive Shock:

An acute obstruction to ventricular outflow tract can cause obstructive shock. Causes include pulmonary embolus, tension pneumothorax, cardiac tamponade and obstructive lesions of the left heart (hypertrophic left heart syndrome, coarctation of aorta, critical aortic valve stenosis and interrupted aortic arch).An acute raise in SVR results from a sudden reduction in cardiac output and functional hypovolemia. Rapid identification of the cause of obstructive shock is significant in acute management [11,12].

Distributive Shock:

It is due to inappropriate vasodilatation and pooling of blood in the peripheral vasculature. Common causes in children include sepsis, anaphylaxis and drug ingestions.

Pediatric sepsis is defined as the systemic inflammatory response syndrome which includes either an abnormal temperature or leukocyte count along with tachycardia/bradycardia and/or tachypnea, in presence of a confirmed or suspected invasive infection [10,12].

Severe sepsis is defined as sepsis plus 1) Cardiovascular dysfunction 2) Acute respiratory distress syndrome or 3) > 2 organ system dysfunctions[10,12].

Although most patients who present with sepsis, severe sepsis or septic shock have negative cultures, exposure to microbial components triggers an inflammatory cascade along with activation of coagulation and vascular mediators that results in severe capillary leak, myocardial depression and vasomotor instability[12]. Although classically septic shock results in hyperdynamic circulation and low SVR that manifest as **warm shock** many children with septic shock present with low cardiac output and elevated SVR or **cold shock**.

Various studies have attempted to find prognostic indicators for septic shock. In adults elevated levels of Inteleukin 1 B and tumour necrosis factor are associated with a poor outcome. In pediatric septic shock there is higher levels of interleukin -6 and erythropoietin. CRP levels more then 8mg/dl is 94% sensitive and 87% specific for SIRS. **Procalcitonin** is another very specific marker for children with septic shock and correlates with higher mortality [11,12].

The most studied prognostic marker for severity of septic shock is blood lactate. Normalization of blood lactate and base deficit has been associated with recovery from septic shock[10,11,12].

In anaphylaxis, the release of preformed histamine, proteases and proteoglycans in mast cells followed by prostaglandins and leukotrienes leads to classic symptoms of the skin and respiratory tract as well as a profound vasodilatory response resulting in a low SVR along with capillary leak causing hypovolemia [11].

Neurogenic Shock:

It is one of the causes of distributive shock resulting from sudden loss of sympathetic nerve supply to the vascular smooth vessel leading to profound decrease in the SVR. Unlike in other types of shock the unopposed vagal activity classically results in bradycardia and absence of classical tachycardia response to hypotension[11].

Cardiac output is controlled by both the heart rate and stroke volume of each contraction. Preload, after load and the cardiac contractility determine stroke volume.

Preload is an indicator of both the absolute intravascular volume and also changes in venous capacity. Afterload is related to Systemic vascular resistance.

Primary response to compensate for shock is through the sympathetic nervous system. Baroreceptors in the carotid sinus and the chemoreceptors in the aorta and the carotid bodies sense the hypovolemia and hypoxemia and inturn activate the sympathetic adrenergic response. The release of catecholamines activate alpha1 receptors in the peripheral vascular smooth muscles and cause peripheral vasoconstriction. Presynaptic α2 receptors in the vasculature and the heart are activated by norepinephrine which is released from sympathetic nerves and mediate the negative feedback inhibition of further norepinephrine release [11,12].

Post synaptic α1 and α2 receptors in the peripheral vessels mediate vasoconstriction. In the heart β1 receptors are activated to increase heart rate (chronotrophy), conduction velocity(dromotrophy) and contractility(inotropy) [12].

In heart failure β2 receptors are activated and cause negative inotropic effects mediated by nitric oxide and cyclic GMP. Peripheral Dopamine(D1) receptors mediate dilatation of the coronary and mesenteric arteries and stimulate natriuretic response [12].

Phosphodiesterase inhibitors increase the contractility by increasing the cyclic AMP and potentiating the delivery of calcium into myocardial cells. At the same time, PDE inhibitors dilate peripheral blood vessels.

In addition to sympathetic nervous system endocrine mediated mechanisms help maintain perfusion in shock. Atrial receptors in the heart sense decreased volume and stimulate the release of antidiuretic hormone which acts as a peripheral vasoconstrictor and increases water reabsorption at the renal collecting ducts. The

RAAS is activated in shock when the kidney release renin in response to decreased renal blood flow. Angiotensin II is a peripheral vasoconstrictor and aldosterone will increase volume via sodium reabsorption at the kidneys[11,12].

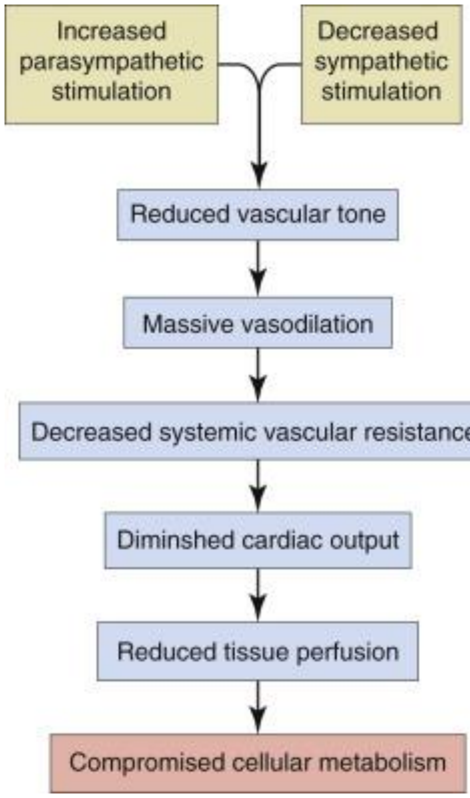


Figure 4: Pathophysiology of Neurogenic shock[10]

Stages of Shock:

1) **Compensated Shock:** In this stage normal circulatory compensatory mechanisms eventually cause full recovery.
2) **Decompensated Shock:** In this stage without therapy the shock becomes steadily worse until death.
3) **Irreversible Shock:** In this stage the shock has progressed to such an extent that all forms of known therapy are inadequate to save the patient's life.

Nonprogressive Shock – Compensated Shock:

If the shock is not severe enough to cause its own progression the person eventually recovers. Therefore shock is of lesser degree and is called non progressive shock, meaning that the sympathetic reflexes and other factors compensate enough to prevent further deterioration of the circulation[11].

The factors that cause a child to recover from moderate degree of shock are all negative feedback control mechanisms of the circulation which attempt to return cardiac output and arterial pressure back to normal [12]. They include:

1) Baroreceptor reflexes which elicits sympathetic stimulation of the circulation.
2) CNS ischemic response: which elicits even more powerful sympathetic stimulation throughout the body.
3) Increased secretion of renin by the kidneys and formation of angiotensin II, which constricts the peripheral arteries and also causes decreased output of water and salt by kidneys.

4) Increased secretion by the posterior pituitary gland of vasopressin which constricts the peripheral arteries and veins and greatly increases water retention by the kidneys.
5) Increased secretion by the adrenal medulla of epinephrine and norepinephrine
6) Compensatory mechanisms that return the blood volume back towards normal including absorption of large quantities of fluid from the intestinal tract, absorption of fluid into the blood capillaries from the interstitial spaces of the body, conservation of water and salt by the kidneys and increased thirst and increased appetite for salt

Progressive Shock:

It is caused by a vicious cycle of cardiovascular deterioration.

Cardiac Depression: When the arterial pressure falls low enough, coronary blood flow decreases below that required for adequate nutrition of the myocardium. This weakens heart muscle and thereby decreases the cardiac output more. Thus a positive feedback cycle has developed whereby the shock becomes more and more severe. Thus, one of the important features of progressive shock whether it is hemorrhagic in origin or caused in any other way is eventual progressive deterioration of the heart [11,12].

Vasomotor Failure: In early stages of shock, various circulatory reflexes cause intense activation of the sympathetic nervous system. This helps delay depression of cardiac output and helps prevents decreased arterial pressure. However there comes a point when diminished blood flow to the brain vasomotor center depresses the center so much that it too becomes progressively less active and eventually inactive [12].

Blockage of very small vessels: In time blockage occurs in many of the very small blood vessels in the circulatory system and this also causes progression of shock. The initiating cause is the sluggish blood flow in the microvessels. Because tissue metabolism continues despite the low flow, large amounts of acid both lactic acid and carbonic acid continue to empty into the local blood vessels and greatly increase the blood acidity. This acid plus other deterioration products from the ischemic tissues causes local blood agglutination resulting in minute blood clots leading to very small plugs in the blood vessels [12].

Increased Capillary permeability: After many hours of capillary hypoxia and lack of other nutrients the permeability of the capillary blood vessels increases and large quantities of fluid begin to transudate into the tissues. This decreases the blood volume even more with a resultant further decrease in cardiac output making shock still worse [11,12].

Release of toxins from ischemic tissues and cardiac depression is caused by the release of these toxins.

Generalised cellular deterioration: As shock becomes more severe many signs of generalized cellular deterioration occur throughout the body. One organ especially affected is the liver which occurs because of lack of enough nutrients to support the normally high rate of metabolism in liver cells. There is further deterioration of many organs including the lung (with eventual development of pulmonary edema and poor ability to oxygenate the blood) and the heart [12].

IRREVERSIBLE SHOCK

After shock has progressed to a certain stage, transfusion or any type of therapy becomes futile in saving the child's life [12].

Sometimes even in this stage of irreversible stage therapy on rare occasions can return the arterial pressure and the cardiac output but only for short periods of time, and the circulatory system continues to deteriorate and death ensues sooner or later [12].

There are multiple deteriorative changes that occurs in the muscles of the heart that may not necessarily affect the heart's immediate ability to pump blood but over a longer period of time they depress the heart's pumping mechanism to cause death. Beyond a certain point so much tissue damage has occurred, so many destructive enzymes have been released into the body fluids, so much acidosis has developed and so many other destructive factors are now in progress that even a normal cardiac output for a few minutes cannot reverse the continuing deterioration [11,12].

Thus, in severe shock a stage is reached at which the person will die even though vigorous therapy might still return the cardiac output to normal for short periods of time.

DEPLETION OF HIGH ENERGY PHOSPHATE RESERVES IN IRREVERSIBLE SHOCK:

The high energy phosphate reserves in the tissues of the body especially in the liver and the heart are greatly diminished in severe degrees of shock. Essentially all the creatinine phosphate has been degraded and almost all the ATP has been downgraded to ADP and adenosine [11,12].

Then much of this adenosine diffuses out of the cells into the circulating blood and is converted to uric acid, a substance that cannot reenter into the cell to reconstitute the adenosine phosphate system.

New adenosine can be synthesized at a rate of only about 2 % of the normal cellular amount an hour meaning that once the high energy phosphate stores of the cells are depleted, they are difficult to replenish [12].

Thus, one of the most devastating end results of deterioration in shock and one that is perhaps most significant for development of final state of **irreversible shock is this cellular depletion of these high energy stores.**

PRINCIPLES OF MANAGEMENT OF PEDIATRIC SHOCK

Early goal-directed therapy (EGDT) is targeted at maintaining and restoring airway, oxygenation, ventilation, and circulation within initial first hour of shock onset. Adequate circulation is further defined by adequate perfusion, normal blood pressure for age, and normal or threshold heart rate [13,14].

Appropriate therapeutic goals for the treatment of pediatric shock should include the following [13,14]:

- Normal mental status
- Normal blood pressure for age
- Normal or threshold heart rate for age
- Normal and equal central and peripheral pulses
- Warm extremities with capillary refill of 2 seconds or less

- Urine output greater than 1 mL/kg/h
- Normal serum glucose levels
- Normal serum ionized calcium levels
- Decreasing serum lactate levels

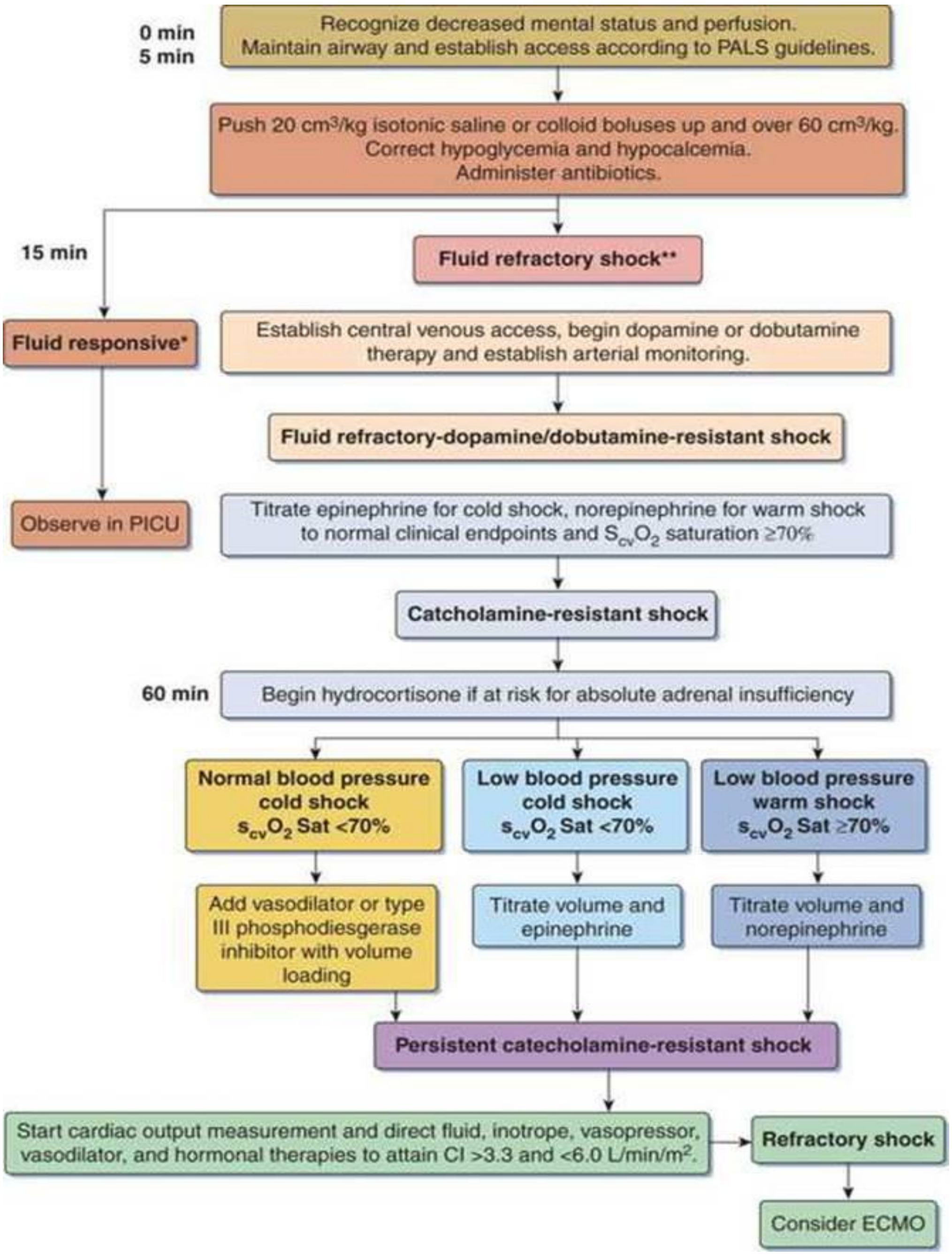


Figure 5: Management Protocol for shock in paediatrics [13].

Initial Resuscitation

Independent of etiology of shock, ABCs (airway, breathing, circulation) must be evaluated and stabilized without further delay involving diagnostic workup or imaging studies [13,15]. The patients should be placed in appropriate position for non-invasive monitors for pulse rate and oxygen saturation monitoring.

Patient's airway should be patent, and patient must be adequately oxygenated and ventilated. Initially, provide 100% supplemental O2 at a high flow rate through a face mask or, in case respiratory distress is present, through non-invasive continuous positive airway pressure (CPAP) or high flow nasal cannula. In case the patient is in respiratory failure, immediate ET intubation should be followed and patient should be put on mechanical ventilation. In cases where airway can be maintained and oxygenation supported without immediate intervention, it is better to delay intubation so that aggressive fluid resuscitation can be done. This is recommended considering the negative (and potentially catastrophic) effect of PPV on venous return and cardiac stability in the hypovolemic patient [13,14].

Once airway has been stabilized and adequate ventilation and administration of oxygen have been ensured, immediate attention should be given to improving circulation and oxygen delivery to the vital organs. Circulatory improvement is achieved by volume expansion and, in cases of fluid refractory shock, pharmacologic therapy with vasopressors and cardiac inotropic agents is used [13,14,15].

Glucose and Calcium Stabilization

Children in shock, are at risk for both hypoglycemia and hypocalcemia. Both of these conditions need to be rapidly identified and corrected [15,16].

Hypoglycemia

Hypoglycemia is common due to many reasons, including of inadequate glycogen stores, metabolic failure and increased consumption. High levels of endogenous and exogenous catecholamines leads to a relative insulin-resistant state which can inturn result in serum hyperglycemia [14,15].

In all children presenting with shock an immediate bed side glucose should be tested. In case of hypoglycemia, the child is immediately provided with intravenous dextrose. The dose of dextrose is 0.5-1 g/kg. It can be administered as follows[14,15]:

- 5-10 mL/kg of D10W
- 2-4 mL/kg of D25W
- 1-2 mL/kg D50W

Hypocalcemia

Hypocalcemia in patients presenting with shock will be due to impaired parathyroid hormone function, decreased hepatorenal vitamin D hydroxylation, and end-organ resistance[16].

Calcium is an important factor which mediates excitation and contraction coupling in muscle cells, including cardiac muscle; low levels of ionized calcium lead to cardiac dysfunction and severe organ dysfunction. The availability of ionized calcium depends on the patient's serum acid-base status; an acidic environment leads to dissociation of calcium from proteins, making it available as a cofactor in cell function[14,16].

Calcium is administered either as calcium gluconate or as calcium chloride. However, calcium chloride is recommended as it has been shown to produce higher levels of available calcium and, hence, is recommended in acute resuscitation of a child in shock. The recommended dose of calcium chloride (10%) is 10-20 mg/kg (0.1-0.2 mL/kg) IV, administered as an IV infusion rate and should not exceed 100 mg/min. Calcium should never be given empirically during active CPR without any clear indication because this has been associated with increased mortality[16].

Fluid Resuscitation

The major physiologic abnormality which is seen in most forms of pediatric shock is intravascular hypovolemia. It has been proven that children with hypovolemic shock who receive adequate, appropriate aggressive fluid management during the first hour of resuscitation have higher chances of survival and recovery [13,14].

Immediate vascular access should be secured and if possible, a minimum of two large bored free flowing IV catheter should be secured. In case a vascular access is not easily achieved, then an intraosseous needle should be placed into the bone marrow for rapid fluid resuscitation [14,15]. An IO line is considered as good as an IV line for the purpose of fluid or medication administration that is necessary for the acute resuscitation of a compromised infant or child in shock [16,17].

20 mL/kg of an isotonic crystalloid fluid infusion, such as 0.9% sodium chloride or Ringer lactate solution is administered over 5 minutes or less. This is rapidly achieved by disconnect-reconnect technique using large volume syringes. In this technique, one provider prepares syringes of normal saline or lactated Ringer solution while the other pushes the fluid filled syringe into an IV or IO catheter.

Soon after initial bolus of fluid (20 mL/kg) has been administered, reassess the patient. In case the patient has persistent shock, immediately infuse next 20 mL/kg of fluid and repeat the cycle[18,19]. Additional boluses are titrated depending on the clinical improvement of the child along with improved mental status, perfusion, hemodynamic stability, and urinary output. A child with severe hypovolemia or sepsis should receive 60 mL/kg of volume in initial 15 minutes of early goal-directed therapy (EGDT)[14,15,20].

In case where more than 2-3 volumes of crystalloid boluses have been infused into a patient with hemorrhage (eg. from trauma) then blood or PRBC transfusion should be given. During fluid resuscitation child should be assessed for basal crepitations and hepatomegaly in cases of which there should be immediate transition from fluid therapy to inotrope therapy [14,15].

In children presenting with cardiogenic shock, judicious fluid boluses of 5-10 mL/kg should be given and balanced with potential need for early inotropic support to prevent fluid overload.

Vasoactive Agents

If shock persists even after the initial fluid resuscitation, then it is described as fluid-refractory shock. In such cases early initiation of inotropic catecholamine infusions is recommended to help restore total arterial flow of oxygen by improved perfusion and cardiac function [14,21].

Initially the inotropes can be administered through the peripheral line till the central line is secured. The inotropic agent which has to be used depends on the cardiac output, systemic vascular resistance (SVR and the overall hemodynamics of the patient in shock. For a child in cold shock with elevated systemic vascular resistance, dopamine and epinephrine are first-line agents. For a child presenting with warm shock and low SVR, norepinephrine is recommended. Based on clinical measures to include the central venous oxygen saturation ($ScvO_2$) and potentially directly or indirectly measured cardiac index (CI), vasodilators such as milrinone may also be used to treat low cardiac output states [14,15].

In cases of fluid and inotrope resistant shock other conditions should be considered. Pericardial effusion, pneumothorax, and pulmonary embolism all should be ruled out. Malignant arrhythmias if any should be converted to normal sinus rhythm as soon as possible. Consideration of a potential endocrine emergency, such as relative/absolute adrenal insufficiency or hypothyroidism is also necessary [22,23].

Antibiotics and Source Control

In case of septic shock, initial coverage with empirical antibiotics is essential in order eliminate the precipitating cause of shock. The current standard of care is to initiate empirical antibiotics in first hour of diagnosis of severe sepsis. Delay in antimicrobial therapy, greater than 3 hours after recognition of sepsis, has been associated with increased mortality and prolonged organ dysfunction [14,22].

Blood cultures should be obtained before antibiotic administration if possible, or as soon as clinical stability permits. Early source control is also recommended [22].

Supportive Therapies

Corticosteroids

The use of corticosteroids in shock, particularly septic shock, is controversial. Many large-scale, controlled trials in animals and humans have not demonstrated improved outcome with corticosteroid use. Nevertheless, a question remains as to whether patients in severe septic shock have adequate levels of circulating glucocorticoids to support their physiology when it is severely stressed[14,24].

In a secondary analysis of 288 previously published pediatric subjects with septic shock, Wong et al combined prognostic and predictive enrichment strategies to identify a pediatric septic shock subgroup responsive to corticosteroids. They found evidence that a combination of prognostic and predictive strategies based on serum protein and messenger RNA biomarkers can identify a subgroup of children with septic shock who may be more likely to benefit from corticosteroid treatment. In a subgroup of children who were at intermediate to high pediatric sepsis biomarker risk model-based risk of mortality, investigators found that corticosteroids were independently associated with more than a 10-fold reduction in the risk of a complicated course (relative risk, 0.09; 95% CI, 0.01-0.54; p = 0.007). [14,24]

Adrenocortical failure or infarction, known as Waterhouse-Friderichsen syndrome, may result in cardiovascular failure and hyporesponsiveness to catecholamines. In affected patients, initiation of stress-dose hydrocortisone, in the range of 50-100 mg/m^2/day IV, may be beneficial and lifesaving. A serum cortisol level may be drawn prior to initiating the first dose of corticosteroids, and if this random serum cortisol level is low, then replacement doses may be beneficial. Moreover, some data suggest a potential role for corticosteroid replacement therapy in select patients with septic shock[14,15].

Furthermore, select patients may have adrenal insufficiency, rendering them fluid refractory and catecholamine resistant during resuscitation from shock. Some practitioners evaluate a baseline serum cortisol level in children with fluid-refractory, catecholamine-resistant shock and/or perform a corticotropin stimulation test with 250 mcg of corticotropin, and then treating the patient with hydrocortisone, although the use of such serum measurements has not been shown to result in improved outcomes[25,26].

Therapy is continued for patients who prove to have an absolute baseline cortisol level of less than 20 mcg/dL and/or a depressed response to the corticotropin stimulation test (ie, a rise of < 9 mcg/dL at 30 and 60 minutes after administration of corticotropin)[14,15,27].

Bicarbonate

Sodium bicarbonate use in the treatment of shock is also controversial. During shock, acidosis develops, which impairs myocardial contractility and optimal function of catecholamines. However, treatment with bicarbonate may worsen intracellular acidosis while it corrects serum acidosis. This occurs because bicarbonate is an ion that does not readily traverse semipermeable cell membranes[14,15]. Hence, bicarbonate combines with acid in serum, resulting in the production of carbon dioxide and water, as defined by the Henderson-Hasselbalch equation[14,15].

If the increased carbon dioxide is not removed via ventilation, it readily enters the cell and drives the Henderson-Hasselbalch reaction in the opposite direction, thereby increasing intracellular acidosis. Worsened myocardial intracellular acidosis may result in a decrease in myocardial contractility. In addition, bicarbonate administration may result in hypernatremia and hyperosmolality, thereby decreasing the availability of ionized calcium[14,15].

Finally, laboratory and clinical data have not demonstrated that bicarbonate administration improves the ability to defibrillate, improves total arterial flow of oxygen (DO_2), or improves survival rates in shock and cardiac arrest. Studies in patients with cardiovascular arrest have not demonstrated improved survival rates associated with the use of bicarbonate. Thus, acidosis that results from shock should ideally be corrected with increased perfusion from volume supplementation and judicious use of inotropic medications in conjunction with optimal ventilation[14,15].

Medication Summary

A variety of therapeutic treatments may be required for pediatric patients in shock. Possible therapies may include the following:

- Inotropic medications
- Dextrose
- Electrolytes and calcium stabilization
- Prostaglandin E1
- Corticosteroids

Inotropic agents

Class Summary

Inotropic agents increase myocardial contractility and have variable effects on peripheral vascular resistance[11]. First-line inotropic agents in pediatric shock include dopamine and epinephrine. On the basis of cardiac output (CO) and systemic vascular resistance (SVR), other vasoactive agents indicated in the treatment of shock may include vasoconstrictors (eg, norepinephrine, phenylephrine) or vasodilators (eg, dobutamine, milrinone). Which agents are indicated and which are effective in patients with any given etiology of shock depends on the end-diastolic volume and contractile state of the patient's cardiovascular system[14,15].

Dopamine

Dopamine is used for refractory hypotension following adequate volume resuscitation. It stimulates beta1- and alpha1-adrenergic and dopaminergic receptors in a dose-dependent fashion. In low doses, dopamine acts on dopaminergic receptors in renal and splanchnic vascular beds, causing vasodilatation in these beds. In midrange doses, it acts on beta-adrenergic receptors to increase heart rate and contractility, improve cardiac output, and enhance conduction (increasing sinoatrial rate) in the heart. In high doses, it acts on alpha-adrenergic receptors to increase systemic vascular resistance and raise blood pressure[14,15].

Dobutamine

Sympathomimetic agent with primarily beta1-agonist effects, increasing heart rate and blood pressure. Some weak beta2-mediated peripheral vasodilation. Little effect on alpha receptors. May precipitate ventricular dysrhythmias, although potentially less likely to do so than epinephrine[14,15].

Epinephrine (Adrenalin)

Epinephrine is used for hypotension refractory to dopamine. Its alpha-agonist effects include increased peripheral vascular resistance, reversed peripheral vasodilatation, systemic hypotension, and vascular permeability. Its beta2-agonist effects include bronchodilation, chronotropic cardiac activity, and positive inotropic effects[14,15].

Norepinephrine

Norepinephrine is used for protracted hypotension following adequate fluid-volume replacement. This agent stimulates beta1- and alpha-adrenergic receptors, thereby increasing cardiac muscle contractility and heart rate, as well as vasoconstriction [14,15].

Phenylephrine

Strong alpha-receptor stimulant with little beta-adrenergic activity that produces vasoconstriction of arterioles in the body, helping increase systemic vascular resistance[14,15].

Phosphodiesterase Enzyme Inhibitor

Class Summary

Milrinone is the primary phosphodiesterase (PDE) inhibitor used in pediatrics, and it works via a different mechanism than that of the catecholamines. Milrinone inhibits PDE III, producing an increase in intracellular cyclic adenosine monophosphate (cAMP), which raises intracellular calcium levels and thereby improving cardiac inotropy and peripheral vasodilation[16,17].

Milrinone may be used together with catecholamines to further increase myocardial contractility while reducing systemic vascular resistance (SVR) and afterload. It can be useful in improving perfusion in patients who remain in compensated shock with poor peripheral perfusion but a normal central blood pressure and adequate intravascular volume. Milrinone is also often a useful adjunct in patients who have low cardiac output syndrome following congenital heart disease corrective surgeries[14,15,16].

Adverse effects of milrinone may include arrhythmias and thrombocytopenia. [Care must be taken when choosing to start phosphodiesterase inhibitors because of their vasodilator effects and their long half-life[16,17].

Milrinone

Milrinone is a selective PDE III inhibitor that acts as a positive inotrope and vasodilator with little chronotropic activity[15].

BIOMARKERS USED IN PREDICTING THE PROGNOSIS IN PEDIATRIC SEPTIC SHOCK.

Table 1:Biomarkers used in predicting the prognosis in pediatric septic shock[14]

Biomarkers currently used in clinical setting	**Information**
Lactate	Prognosis, possible therapeutic decisions?
C-reactive protein	Diagnosis, antibiotic guide in neonates
Procalcitonin	Diagnosis, prognosis, antibiotic guide
Cytokines (interleukin-6, interleukin-8)	Diagnosis, prognosis
Biomarkers currently used in research setting (examples) for potential clinical use	
Neutrophil and monocyte markers (CD 64, CD 163, CD 11b, CD 15s)	Diagnosis
Triggering receptor expressed on myeloid cells-1	Diagnosis
Cytokines (tumor necrosis factor-alpha, interleukin-18)	Diagnosis, prognosis
Lipopolysaccharide-binding protein	Diagnosis
Vasoactive peptides (pro-adrenomedullin, pro-endothelin, pro-atrial natriuretic peptide, copeptin, etc.)	Prognosis
Endothelial/leukocyte adhesion molecules [soluble intercellular adhesion molecule (ICAM)-1, vascular cell adhesion molecule (VCAM)-1, E-selectin]	Prognosis

Biomarkers currently used in clinical setting	Information
PERSEVERE model [1] candidate biomarkers: C–C chemokine ligand 3 and 4 (MIP-1alpha) Neutrophil elastase 1 Fibrinogen-like 2 Granzyme B Heat-shock protein 70 kDa 1B IL-1α IL-8 Lipocalin 2 (NGAL) Lactotransferrin Matrix metallopeptidase 8 Orosomucoid 1 Resistin Sulfatase 2 Thrombospondin 1	Prognosis

Serum Lactate

Background and History

Lactic acid is a carbohydrate within cellular metabolism and its levels increases with increase in metabolism during exercise and with catecholamine stimulation. Glucse-6-phospate is converted by anerobic pathway to pyruvate via the Embden-Meyerhof pathway. Pyruvate and lactate are in a state of equilibrium with a ratio of about 25 lactate to 1 pyruvate molecules. Thus, lactate is the normal byproduct of the anaerobic metabolism of glucose in the tissues. The lactate thus formed exits the cells and is carried to the liver, where it undergoes oxidization back to pyruvate and converted to glucose through the Cori cycle [28]. All tissues in our body can utilize lactate as a source of energy, as it is converted back to pyruvate quickly and enters the **Krebs cycle**. In cases of decreased tissue oxygenation, pyruvate is not metabolized and leads to raise in the intracellular levels, leading to increase in lactate levels proportionately. With a chronic oxygen debt and decompensation of the body's buffering abilities (either from long-term dysfunction or from excessive production), hyperlacticaemia and metabolic acidosis follow, commonly known as lactic acidosis [2,3,28].

Lactic acid exists in two isomeric forms, L-lactate and D-lactate.

L-lactate is one which is most commonly measured, as it is the only form which is produced in human metabolism. Raise in its level represents increased anaerobic metabolism due to **tissue hypoperfusion**.

During the early period of the 20th century, many physicians found out that patients who are critically ill will have metabolic acidosis not associated with elevation of ketones or any other measurable anions[28]. In 1925, Clausen discovered accumulation of lactate in blood as a main cause of acid-base disorder. Many decades later, Huckabee's work firmly established that lactic acidosis accompanies many severe illnesses and pathogenesis is due to tissue hypoperfusion [28].

The causes of lactic acidosis are enlisted below in Fig 1.

Pathophysiological classification of lactic acidosis

HYPOXIC	NON-HYPOXIC
Ischemia	**Delayed Clearance**
Shock, severe anemia, cardiac arrest	Renal or hepatic dysfunction
Global Hypoxia	**Pyruvate Dehydrogenase Dysfunction**
Carbon monoxide poisoning	Sepsis, thiamine deficiency, catecholamine excess, alcoholic and diabetic ketoacidosis
Respiratory Failure	**Uncoupling of Oxidative Phosphorylation**
Severe asthma, COPD, asphyxia	Cyanide, salicylates, methanol & ethylene glycol metabolites, anti-retroviral drugs, valproic acid, biguanides, INH
Regional Hypoperfusion	**Accelerated Aerobic Glycolysis**
Limb or mesenteric ischemia	Increased effort, sepsis, seizures, large fructose loads, malignancies

Figure 6. Causes of lactic acidosis[28]

The normal range of blood lactate concentration is 0.5-1 mmol/L[28]. In patients who are critically ill lactate concentrations less than 2mmol/l is considered normal. Hyperlactatemia is defined, as lactate levels of more than 2mmol/l. Serum lactate levels of 2-4mmol/l is considered as mild to moderate hyperlactatemia. More than 5 mmol/l is considered as lactic acidosis [28,29].

Hyperlactatemia can occur in case of proper tissue perfusion, intact buffering systems, and sufficient tissue oxygenation [28].

Lactic acidosis, will be associated with severe metabolic dysregulation, tissue hypoperfusion and inborn errors of metabolism.

Lactic acidosis may not produce acidemia in a patient. Development of lactic acidosis depends on range of hyperlactatemia, the buffering ability of the body, and the existence of other conditions that result in tachypnea and alkalosis (eg, liver disease, sepsis). Hence, hyperlactatemia or lactic acidosis may be present along with acidemia, a normal pH, or alkalemia [28].

Many etiologies may be responsible for lactic acidosis, most common are circulatory failure and hypoxia. Evidence shows that increased morbidity and mortality is seen in patients with either persistently elevated or increasing lactate levels [28,29]

Metabolic acidosis

Metabolic acidosis can be defined as decreased systemic pH either due to a primary increase in hydrogen ion (H^+) or due to reduction in bicarbonate (HCO_{3-}) concentrations. In the acute state, respiratory compensation of acidosis occurs by mechanism of hyperventilation thus resulting in a reduction in $PaCO_2$. Chronically, renal compensation occurs by reabsorption of HCO_3 [28,29].

Acidosis arises from an elevated production of acids, a reduction of alkali, or a reduced renal excretion of acids. The etiology of metabolic acidosis is classified as into those that have an elevated anion gap and those that do not. Lactic acidosis, determined by a state of acidemia and a elevated serum lactate concentration, is one type of anion gap metabolic acidosis and may result from numerous conditions [28].

Severe metabolic acidosis with arterial blood pH of less than 7.2 is associated with impaired cardiac contractility and suboptimal response to exogenous catecholamines. It has been demonstrated that elevated serum lactate has negative inotropic effects which are independent of serum pH.

Types of lactic acidosis

Cohen and Woods classified lactic acidosis into 2types, type A and type B[28]

Type A is lactic acidosis that occurs with clinical signs of tissue hypoperfusion or poor oxygenation of tissues (eg, hypotension, cyanosis, cool and mottled extremities). It can occur either by the overproduction of lactate or by the underutilization of lactate. In case of overproduction, pulmonary, circulatory and haemoglobin transfer disorders will be most likely etiology[30].

In cases of underutilization of lactate gluconeogenesis inhibition, thiamine deficiency, and liver disease can be responsible.

Type B is lactic acidosis occurring when no clinical evidence of poor tissue perfusion or oxygenation exists. However, in many cases of type B lactic acidosis, occult tissue hypoperfusion is now recognized to accompany the primary etiology [28,29].

Type B is divided into 3 sub categories depending on the underlying etiology [28,29].

Type B1 occurs in systemic disease, such as kidney and liverfailure, diabetes mellitus and malignancy.

Type B2 occurs in several classes of drugs and toxins, including biguanides, alcohols, iron, isoniazid, zidovudine, and salicylates [28].

Type B3 occurs due to IEM.

Shock may move from type A to type B, as in the beginning there is often hypoperfusion, and later with aggressive fluid resuscitation hypoperfusion resolves, but lactic acidosis still persists due to altered oxidative phosphorylation and leukocyte production of lactate[31].

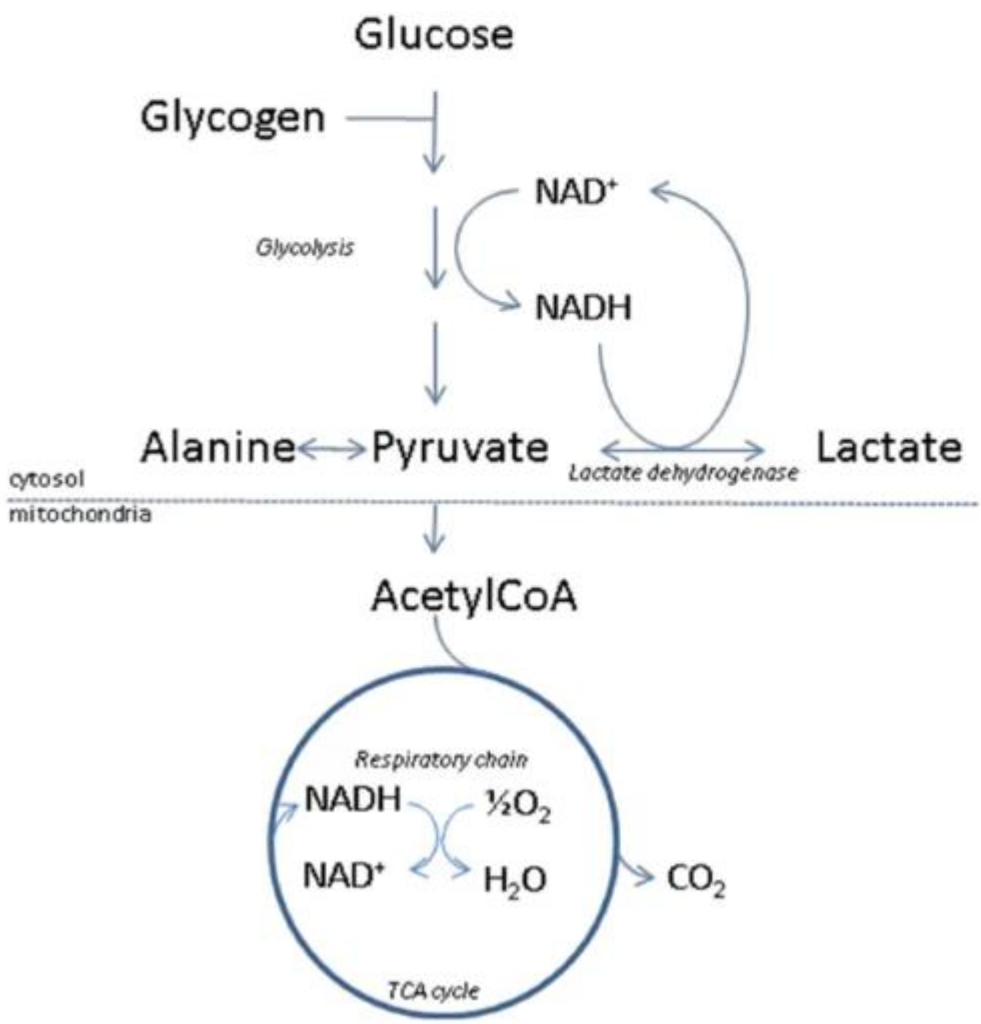
Mechanism of Lactate production

The anaerobic metabolic pathway also known as glycolysis is the initial step of glucose metabolism and occurs in virtually all cells. The by product of this pathway is pyruvate, which later diffuses into the mitochondria and will be metabolized into carbon dioxide by another, metabolic pathway, the Krebs cycle. The metabolism of glucose into pyruvate also leads to chemical reduction of the enzyme cofactor oxidized form nicotinic acid dehydrogenase (NAD^+) to nicotinic acid dehydrogenase (NADH) (reduced form) [28].

Erythrocytes have the capacity of carrying out glycolysis; however, erythrocytes do not have mitochondria hence cannot use oxygen to generate adenosine triphosphate (ATP). The pyruvate formed in glycolysis is metabolized through the enzyme lactate dehydrogenase (LDH) to lactate. The anaerobic pathway is not efficient, and only 2 molecules of ATP are generated for every molecule of glucose which is converted to lactate. Lactate thus produced diffuses out of the cells and is converted into pyruvate and is later aerobically metabolized into carbon dioxide and ATP[30].

Alternatively, liver and kidney can use lactate to generate glucose through another pathway known as gluconeogenesis. Metabolism of glucose to lactate in one tissue, and conversion of lactate to glucose by another tissue, is termed the **Cori cycle**[31,32].

Lactate is cleared from blood, mainly by the hepatic cells, with the renal tissue (10-20%) and skeletal muscles to a lesser extent. Ability of the liver to clear lactate is dependent on its concentration and progressively decreases as the level of blood lactate raises. Lactate clearance by the liver is impaired by several other factors, such as acidosis, hypoperfusion, and hypoxia.



Outline of lactate metabolism. With insufficient oxygen supply, pyruvate will be diverted to lactate, thereby assuring regeneration of NAD^+ from NADH. This will enable glycolysis, and the accompanying ATP production to proceed.

Figure 7: Lactate metabolism[26].

Metabolic aspects of lactate production

Normal serum lactate concentration is below 2 mmol/L [28,29], lactate turnover in resting and healthy humans is approximately 1300 mmol every 24 hours. Lactate producers include skeletal muscle, the gut, the brain and the RBC's. Lactate metabolizers are the hepatic cells, the renal, and the heart. When lactate blood levels exceed 4 mmol/L, the skeletal muscle becomes a net consumer of lactate.

Lactate is an end product of glycolysis; which is formed in the cytoplasm catalyzed by the enzyme lactate dehydrogenase:

Pyruvate + NADH + H^+ = lactate + NAD^+

This is a reversible reaction that favors lactate synthesis with the lactate-to-pyruvate ratio that is normally at 25:1. Lactate synthesis raises when the rate of pyruvate production in the cytoplasm exceeds its rate of clearance by the mitochondria. This occurs when oxygen delivery to the mitochondria declines, such as in tissue hypoxia [31].

Cellular energy metabolism and lactate production Cells requires a supply of energy for synthesis of protein. This energy will be stored in the phosphate bonds of the ATP molecule. Hydrolysis of ATP results in following reaction, where ADP is adenosine diphosphate and Pi is inorganic phosphate[28,29 31]:

ATP = ADP + Pi + H^+ + energy

When there is sufficient supply of oxygen, the cells use ADP, Pi, and H^+ in the mitochondria of cell to reconstitute ATP. During period of cellular hypoxia, the hydrolysis of ATP leads to accumulation of H and Pi in the cytosol. Therefore, ATP hydrolysis is the source of cellular acidosis during hypoxia and not the formation of lactate from glucose, which neither consumes nor generates H^+. The process of glycolysis may be viewed as the following [32]:

D glucose + 2 ADP + 2 Pi = 2 lactates + 2 ATP

The hydrolysis of 2 ATP molecules formed from metabolism of glucose produces H^+, ADP, and Pi, as follows:

2 ATP = 2 ADP + 2 Pi + 2 H^+ + energy

When the oxygen supply is sufficient, the metabolites of ATP are recycled in the mitochondria and the cytosolic lactate concentration increases without acidosis. However, with cellular hypoxia, the equation of anaerobic glycolysis becomes:

D glucose = 2 lactate + 2 H^+ + energy

Cellular transport of lactate

Intracellular accumulation of lactate leads to a concentration gradient favoring its release out of the cell. Lactate leaves the cell and in exchange for a hydroxyl anion (OH-), a membrane-associated, pH-dependent, antiport system. The source of the extracellular OH- ion is the cleavage of water into OH- and H^+. Extracellular H^+ ion combines with lactate leaving the cell, thus forming lactic acid, while intracellular OH- combines with H^+ ion generated during the process hydrolysis of ATP to form water. Therefore, cellular transport of lactate helps to moderate increases in cytosolic H^+ resulting from hydrolysis of anaerobically generated ATP [28,30] .

Etiopathogenesis

Tissue hypoxia leads to more oxygen extraction from the capillary blood. This causes redistribution of the cardiac output to organs as per their ability to recruit capillaries and it also reduces the distance from the capillaries to the cells. With severe reduction in oxygen transport, compensatory raise in the oxygen extraction ratio will be insufficient to continue with aerobic metabolism. Thus, the cell must use anaerobic sources of energy to generate ATP, leading on to generation of lactate and H^+ ions [33,34,35].

The most common cause of lactic acidosis is tissue hypoxia. In ischemic tissues of the skeletal muscle, production of lactate is accelerated along with a concomitant reduction in lactate uptake by the liver, kidney, and myocardium. The accumulation of excess serum lactate overwhelms the body's buffering capacity and results in acidosis.[36,37]

Lactate acidosis as a metabolic monitor of shock

Shock as already discussed can be defined as a clinical syndrome resulting due to an acute imbalance in tissue oxygen requirement and oxygen delivery. Hypoxia in the tissues is the primary problem in most forms of shock. As a result of tissue hypoxia , pyruvate oxidation reduces, lactate production raises, and ATP formation continues through glycolysis pathway. The level of lactate generated correlates with total oxygen debt and also the severity of shock [36].

Serial lactate estimation may be helpful in patients resuscitated from shock to assess the adequacy of therapies.

Hyperlactatemia and Lactic acidosis in sepsis

Patients presenting with severe sepsis or septic shock commonly have hyperlactemia and lactic acidosis[36]. The pathophysiology in cases of sepsis associated lactic acidosis has not yet been understood properly. Increase in the levels of lactate production in anaerobic and aerobic metabolism and decreased lactate clearance leads to hyperlactemia. **Patients presenting with septic shock have lactate levels greater than 4-5 mmol/L, a lactate-to-pyruvate ratio greater more than 10-15:1, and arterial blood pH of less than 7.35 [28,36].**

Even following resuscitation from septic shock, few patients continue to have hyperlactemia (lactate 2-5 mmol/L), in setting of normal blood pH or alkalemia

.These patients have increased oxygen consumption, urea nitrogen excretion in urine, insulin resistance, and a normal lactate-to-pyruvate ratio. Hyperlactemia mostly occurs due to increased production of pyruvate and equilibration with lactate, this has been termed **stress hyperlactemia** [35,36,37].

Several studies have demonstrated the mechanism of lactic acidosis in septic shock is due to an increased lactate-to-pyruvate ratio, showing tissue hypoperfusion and tissue hypoxia as main cause of lactic acidosis [35,36,37]**.**

The additional possible mechanisms include activation of glycolysis and inhibition of pyruvate dehydrogenase. Some studies have demonstrated that patients with sepsis have reduced lactate clearance rather than increased lactate production. Therefore, hyperlactemia may be secondary to raised lactate production in the GIT, hepatic cells, lungs, and skeletal muscles; decreased lactate clearance in the liver; or a combination of both [36].

Hyperlactatemia in patients presenting with sepsis is an indicator of severity of stress response. Hyperlactatemia may develop as a byproduct of overall increase in glycolysis in severe sepsis[37].

Limitations of lactic acidosis as a monitor of tissue perfusion

There are several limitations in using blood lactate as a prognostic indicator in shock. Presence of hepatic failure which is most often present causes a decreased ability for lactate clearance during periods of increased production. Numerous other causes of type B lactate acidosis will lead to hyperlactemia and lactate acidosis in the absence of tissue hypoperfusion. For blood lactate levels to be significantly raised, lactate should be released into the systemic circulation and also the rate of generation must exceed liver, kidney, and skeletal muscle uptake. Hence, regional hypoperfusion of tissues may be present in the setting of normal blood lactate concentrations [33,35,36,].

Diagnostic Considerations[38,39,40]

Conditions to be considered in the diagnosing lactic acidosis :

- Inborn errors of metabolism
- Pyruvate dehydrogenase deficiency
- Oxidative phosphorylation defects
- Cardiogenic shock
- Cardiogenic pulmonary edema
- Pyruvate carboxylase deficiency
- Glucose-6-phosphatase deficiency

Differential Diagnoses[41,42]

- Alcoholic Ketoacidosis
- Anemia
- Bacterial Sepsis
- Distributive Shock
- Haemorrhagic Shock
- Metabolic Acidosis
- Respiratory Failure
- Salicylate Toxicity
- Septic Shock

Serum Lactate Level

The concentration of serum lactate must be analysed as quickly as possible in a sample transported on ice. Normal serum lactate level is less than 2 mmol/L[43,44,45]. Value above 4-5 mmol/L[43,44,45] in setting of acidemia are suggestive of lactic acidosis. In hypoperfused states, persistent hyperlactatemia and lactic acidosis is associated with increased risk of mortality. In case of shock, serial lactate level estimation is useful in knowing the response to therapeutic interventions. Presently, lactate clearance of 10% at 2 hours after initiation of treatment is a proposed method to assess this response. Lactate clearance itself cannot discriminate between oxygen delivery–dependent or oxygen delivery–independent states of hypoperfusion and therefore specific shock treatment (volume resuscitation, inotrope support, vasopressor) cannot be determined from lactate alone [28,31,36].

Discussion On the various mortality scores used in the pediatrics intensive care units

Advances in management protocols in pediatric intensive care units , have resulted in a more sophisticated care for children and adolescents, hence making these units ready to treat cases of high complexity [46,47,48] . Despite these advances, it has been observed the technology available has not always helped and succeeded in improving the quality of patient care and the higher ability to increase life expectancy has become an instrument that could increase the suffering in the child and prolong the death process. Hence, it has become important and necessary to determine about the severity of the disease at admission and assessing its prognosis. This can be done through the mortality prognostic scores that assess the severity of the illness in patients, determining the probability of mortality as per their clinical state and could help in various areas of management and care, such as selection of treatments, economic strategies and ethical issues [49] .

At admission most of the times it is difficult to establish clinical and laboratorial criteria that will allow an estimate of the number and intensity of organ dysfunction and need for therapeutic intervention [46].

Ever since the introduction of mortality scores in the ICU, they have been used more frequently and nowadays the scores are a part of methodology of quality control and research. They are useful for assessing quality of care, prognosis and also to determine the risk of mortality [47,48].

The Pediatric Risk of Mortality score is one of the most important indicators that is used in the pediatric intensive care unit. It was obtained and validated from Physiologic Stability Index (PSI) with 1415 patients evaluated for nine U.S. PICU between 1984 and 1985, and mortality rate was of 116 [46,47,49]. Statistical analysis eliminated the insignificant PSI categories, thus reducing the number of physiological parameters, creating and validating the PRISM. It used 14 parameters (physiological and laboratory data) and used the highest severity value that was recorded in the first 24 hours [48,49]

It presents an excellent discriminatory performance and prediction; it is being used in many pediatric intensive care units as a prognostic score to assess gravity of disease. Some studies show that PRISM score can predict increased risk of mortality while other studies show that PRISM overestimates mortality [48,49].

The Pediatric Risk of mortality score is a second generation physiology base prediction score for mortality in PICU. PRISM was initially derived from PSI , various studies were conducted for the development of PRISM III score which is a third generation score based on the various physiological variable and ranges as well as the diagnostic and other risk variables which are reflective of mortality.

In addition to minimising the period for assessment of mortality risk the predictive performance was maximized while keeping in mind the number of variables and ranges to a minimum extent.

PRISM III uses variables that are readily available and much clearly definable and also maintaining the assumption in the PSI and PRISM that the unmeasured variable is assumed normal [46,49].

Development of PRISM III resulted in many improvements over the original PRISM. The physiological variable and ranges have been revaluated [49] . Predictive power of various physiological variables was reassessed and those that did not contribute significantly to mortality were eliminated. Some variables such as pH, PaO2, creatinine concentration, BUN , WBC and Platelet count have been added [49].

The variables with the greatest importance in outcome prediction were the same in both scores that is low BP, altered mental status and abnormal pupils. While age which was included as an important variable in the original PRISM score has been included in the PRISM III in a logistic form using age appropriate age adjusted physiological variable range [46,49].

Besides PRISM and PRISM III scores another prognostic score which is being used in Pediatric ICU is Pediatric Index of Mortality (PIM). This score was validated in PICU in New Zealand, Australia and United Kingdom [46,18].

Using a practical and objective scoring system to provide clinical and / or laboratorial criteria to evaluate if a delayed treatment is a factor of impact on quality of care in critically ill patients is very important. **The ideal score should be easy to use, does not require extensive experience of the observer, easy to reproduce, low cost, minimally invasive and accurate[5,18,49].**

Variables of PRISM III scores and along with age specific range [46,48,49]

PRISM III Score Parameters

Cardiovascular and neurological vital signs

Systolic blood pressure

Neonate and >55mmhg 0

Neonate and 40-55mmhg 3

Neonate and < 40mmhg 7

Infant and >65mmhg 0

Infant and 45-65mmhg 3

Infant and <45mmhg 7

Child and >75mmhg 0

Child and 55-75 mmhg 3

Child <55mmhg 7

Adolescent and >85mmHg 0

Adolescent and 65-85mmhg 3

Adolescent and <65mmhg 7

Heart rate

Neonate and <215bpm 0

Neonate and 215-225 bpm 3

Neonate and >225 bpm 4

Infant and <215 bpm 0

Infant and 215-225 bpm 3

Infant and >225 bpm 4

Child and <185 bpm 0

Child and 185-205 bpm 3

Child >205bpm 4

Adolescent and <145 bpm 0

Adolescent and 145-155bpm 3

Adolescent and >155bpm 4

Temperature

$<33^{o}$ C 3

33-40^{o}C 0

$>40^{o}$C 3

Mental status

GCS >= 8 0

GCS < 8 5

Pupillary Response

Both reactive 0

1 reactive and 1 fixed 7

Both fixed 11

Acid-Base and Blood gases

Acidosis pH >7.28 and total CO2 >= 17mEq/l 0

pH 7-7.28 or total CO2 5-16.9mEq/l 2

pH <7 or total CO2 <5mEq/l 6

pH

<7.48 0

7.48-7.55 2

>7.55 3

PCO2

<50 mmhg 0

50-75mmhg 1

>75mmhg 3

Total CO2

<=34mEq/l 0

>34 mEq/l 4

PaO2

>50mmHg 0

42-49.9 mmhg 3

<42 mmhg 6

Chemistry Tests

Glucose

<200mg/dl 0

>200mg/dl 2

Potassium

<6.9mEq/dl 0

>6.9mEq/dl 3

Creatinine

Neonate and <0.85 0

Neonate and 0.85 2

Infant <0.9 0

Infant and >0.9 2

Child and <0.9 0

Child and >0.9 2

Adolescent and < 1.3 0

Adolescent >1.3 2

Blood Urea Nitrogen

Neonate and < 11.9mg/dl 0

Neonate and > 11.9 mg/dl 3

Not neonate <14.9mg/dl 0

Not neonate > 14.9 mg/dl 3

Hematologic Tests

WBC count

>3000/cumm 0

<3000/cumm 4

Platelet count

>2lakh/cumm 0

1-2lakh/cumm 2

50000-99000/cum 4

<50000/cumm 5

PT and PTT

Neonate and PT <= 22sec or PTT <=85sec 0

Neonate and PT > 22sec or PTT > 85 sec 3

Not neonate and PT <=22sec or PTT <= 57 sec 0

Not neonate and PT> 22sec or PTT >85 sec 3

Total Prism Score= (Cardiovascular and neurologic subscore)+(acid base and blood gas subscore)+(Chemistry subscore)+(hematologic subscore)

The higher the total score the higher is the mortality

A rising score indicates deterioration

CLINICAL STUDIES

A retrospective study was done by Kim Y A and others on paediatric patients in septic shock at Seoul Korea. In this study a total of 65 patients were included and the risk of mortality of these patients was studied. Serial blood lactate concentration was obtained at time of admission and every 6th hourly after admission upto 24 hours. Parameters which were assessed were the initial blood lactate levels, lactate clearance and a lactate area. These parameters were compared as a predictor of mortality in survivors and non- survivors. Survivors, in comparison with non survivors had an initial lactate concentration of 3.13+/- 2.79 vs 6.16 +/- 4.87 mmol/l ; a lactate clearance of 32.8+/- 63.4 vs -30.8+/- 75.6% and lactate area of 59.7+/- 56.0 vs 168+/-

107 mmol/l. Thus, this concluded that serum lactate and lactate associated parameters can be used as prognostic indicators of mortality in paediatric septic shock patients [1].

A Study was conducted by Kumar N and others on patients admitted to Pediatric Intensive care unit and estimated the serum lactate concentrations initially and after six hours of admission and the clearance was calculated. A negative value indicated an raise in blood lactate.This study included 45 children . The initial lactate concentration was not significantly different between survivors and non survivors [8.44mmol/l vs 7.29mmol/l], however lactate clearance at 6 hours was significantly lower in non survivors compared to survivors . Hence, concluding that lactate clearance and mortality have inverse relationship [4].

A prospective observational study was conducted by Chaudhary J et al on 60 cases of children presenting with septic shock between 1month and 12 years of age over 1 year period. The initial serum lactate values at admission, at 3 hours, at 12 hours and 24 hours were correlated with the mortality of the patients. It was found that a lactate value of > 5mmol/l at 0, 3, 12 and 24 hours of admission to PICU had increased risk of mortality. This study concluded that an increased blood lactate levels at admission is independently associated with higher mortality in children presenting with septic shock [3].

Bai Z and others conducted a prospective observational study on 1109 children admitted to PICU to determine blood lactate levels as a prognostic indicator and a predictor of mortality in critically ill children. Blood lactate achieved an AUC of 0.79 for predicting mortality which is similar to that of PRISM III which achieved a AUC of 0.82. The blood lactate levels had a sensitivity of 61 % and a specificity of 86% in predicting mortality at the optimal cut off value of 5.55mmol/l and the positive and negative likelihood ratios was 4.5 and 0.45 respectively.This study concluded that serum lactate values are equally good as PRISM III scores as predictors of mortality in the critically ill children . They are useful tools in timely intervention in PICU [2].

A prospective observational study was conducted by Jat K R and others in a tertiary care centre. This study was conducted on 30 children presenting with septic shock to PICU. Serum Lactate levels were measured at 0,3,12 and 24 hours of admission. The outcome was correlated with the serum Lactate values. A lactate value of more than 5mmol/l at 0-3,12 and 24 hours of PICU admission had an odds ratio of death of 6.7, 12.5 and 8.6 with a positive predictive value of 38%, 71%, 64%, and a negative predictive value of 80%, 83% and 83% respectively . Hence, this study concluded that non survivors had higher blood lactate levels at admission as well as at 12h and 24h and hence a lactate value of greater than 5mmol/l was a good predictor of mortality [29].

Mudasir Nazim and others conducted a prospective observational study to determine lactate clearance as a prognostic indicator in pediatric septic shock during the first 24 hours of PICU care.112 children were included in this study between the age group of one month and 17 years. Vital parameters, clinical examination, relevant investigations were recorded and PRISM III scores were calculated. Arterial Lactate levels were measured at admission, at 6 hours , 12 hours and 24 hours of admission. Lactate clearance was calculated. They found that lactate clearance at 6 hours and 24 hours was significantly associated with mortality. It was found that there was 24% decrease in mortality for each 10% increase in lactate clearance at 24 hours of admission into the PICU. This study concluded that optimal lactate clearance in pediatric septic shock both during the early presentation and after the initial golden hours is associated with lower mortality. And also 24 hour lactate clearance appears superior to 6 hour lactate clearance in predicting mortality in pediatric septic shock patients [50].

Morris and others conducted a retrospective cohort study in PICU to investigate whether blood lactate concentration on admission predicts mortality in paediatric intensive care and if its addition can improve the performance of the Paediatric Index of Mortality 2 (PIM2) mortality prediction score.A total of 2,380 children were included in this study. Total number of non survivors was 155. It was observed that the admission lactate in non-survivors was higher than in survivors (mean [standard deviation, SD]) 6.6 [5.6] versus 3.0 [2.5] mmol/l, had a positive association with mortality [adjusted odds ratio (OR) for death per unit (mmol/l)] increase 1.11 [95 % confidence interval (CI) 1.06–1.16; $p < 0.001$] and significantly improved the model fit of PIM2 when it replaced absolute base excess. Thus this study concluded that PICU admission blood lactate concentration predicts mortality independently of PIM2[5].

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MATERIALS &

METHODS

MATERIALS AND METHODS-

Source of data: All patients with shock , between the age group of 1 month and 18 years who were admitted to PICU in R L Jalapa hospital and research centre.

Study design: A prospective observational study**.**

Study period: January 2018- May 2019**.**

Inclusion Criteria: All patients with shock , between the age group of 1 month and 18 years who were admitted to PICU in R L Jalapa hospital and research centre.

Shock in any phase that is compensated(Tachycardia, tachypnea, normal or low urine output, normal or slightly elevated BP) decompensated phase(Altered sensorium, cool clammy extremities, blood pressure < -2SD adjusted for age, oliguria, tachycardia, tachypnea) and irreversible shock(comatose, cold cyanotic mottled extremities, not recordable BP, anuria, tachycardia, respiratory failure)

Exclusion Criteria: If the child died within < 2 hours after admission or referred to other hospitals or discharged against medical advice

Sample size: Sample size was estimated based on the study conducted by Dr.Jasashree Chaudhary, Routray SS and Dash LD on the Effectiveness of predicting outcome in critically ill children presenting in septic shock by assessing serum lactate levels [3]. The required sample size was 144.

The formula used to calculate the sample size

$$n = \frac{(r+1)/2\ (z_\alpha + z_{1-\beta})^2\ \sigma^2}{(m_1 - m_2)^2}$$

z_α= Standard normal variable at 95%

$z_{1-\beta}$ = power at 90% = 1.28

σ^2 = variance estimate

r = allocation ratio

m_1 & m_2 are the mean of the 2 groups.

PROCEDURE:

All patients with shock between age group of 1month and 18 years irrespective of aetiology were included in this study after detailed clinical evaluation and diagnosis The patients at admission were evaluated clinically based on the physiologic variables in the PRISM III score and also arterial and venous blood were drawn for routine investigations necessary for evaluating the patients .The clinical status, comorbidities, therapeutic interventions and medications were recorded daily until discharge or death.

Venous sample was obtained without the use of tourniquet or immediately after the tourniquet was removed.

Arterial Blood Gas sample collection:

Patient was placed flat on his back, radial artery was located by performing Allen's test to confirm collateral circulation.

Hand hygiene was performed,impervious gown or apron and face protection was worn. The sampling site on the patient was disinfected with 70% alcohol and allowed to dry.The needle and heparinised syringe was assembled and the syringe plunger pulled to the required fill level recommended by the clinical laboratory.

Blood Lactate was analysed by colorimetric method. A drop of the patients blood was deposited on the slide and evenly distributed by the spreading layer to the underlying layers. The slide incubated and the intensity of the dye complex measured spectrophotometrically.

Principle Of the Procedure:The VITROS LAC Slide method was performed using the VITROS LAC slides and the VITROS chemistry products calibrator kit 1 on VITROS

250/350/950 and 5, 1 FS chemistry systems and the VITROS 5600 INTEGRATED system. The VITROS LAC slide is a multilayered analytical element coated on a polyester support.

Lactate in the sample is oxidized by lactate oxidase to pyruvate and hydrogen peroxide . The hydrogen peroxide generated oxidizes the 3-aminoantipyrene, 1,7-dihydroxynaphthalene dye system in a horseradish-peroxidase-catalysed reaction and results in a dye complex [28-32].

Test type: Colorimetric

Approximate incubation period: 5 minutes.

Temperature :37^{o}

Wavelength: 540 nm[28]

Reaction Sample Volume : 10 microlitres

Measuring range:

Conventional and SI units : 0.5-12mmol/L[28]

Blood lactate levels was estimated at admission and at 12 and 24 hours.

The PRISM III score and S.Lactate values were correlated.

STATISTICAL ANALYSIS:

Data was entered into Microsoft excel data sheet and was analysed using SPSS 22 version software. Categorical data was represented in the form of Frequencies and proportions. **Chi-square test or Fischer's exact test** (for 2x2 tables only) was used as test of significance for qualitative data. Continuous data was represented as mean and standard deviation. **Independent t test** was used as test of significance to identify the mean difference between two quantitative variables. Lactate levels and PRISM III score were further analyzed using the receiver operating characteristic (ROC) and optimal cut-off points were chosen for the calculation of sensitivity, specificity, positive and negative predictive values. A test that predicts an outcome no better than chance has an area under the ROC curve of 0.5. An area under the ROC curve above 0.8 indicated fairly good prediction. The relationship of lactate at admission with the PRISM III score was determined by calculating the Spearman correlation coefficient and two-tailed significance.

Graphical representation of data: MS Excel and MS word was used to obtain various types of graphs

Independent t test was used as test of significance to identify the mean difference between quantitative variables. Lactate levels were analyzed using the ROC curves and optimal cut off points were chosen for the calculation of sensitivity, specificity, positive predictive values and negative predictive values.

P value (Probability that the result is true) of <0.05 was considered as statistically significant after assuming all the rules of statistical tests.

Statistical software: MS Excel**,** SPSS version 22 **(**IBM SPSS Statistics, Somers NY, USA) was used to analyze data

RESULTS

RESULTS

Table 2:- Distribution of subjects according to age group

Age Group	Frequency	Percent
≤1yr	66	45.8
1-5yrs	29	20.1
6-10yrs	16	11.1
>10yrs	33	22.9
Total	144	100.0

Figure 8:- Graph showing Distribution of subjects according to age group

It can be seen that most children belong to the age group of less than 1 year. The children in the adolescent age group are 22.9%.

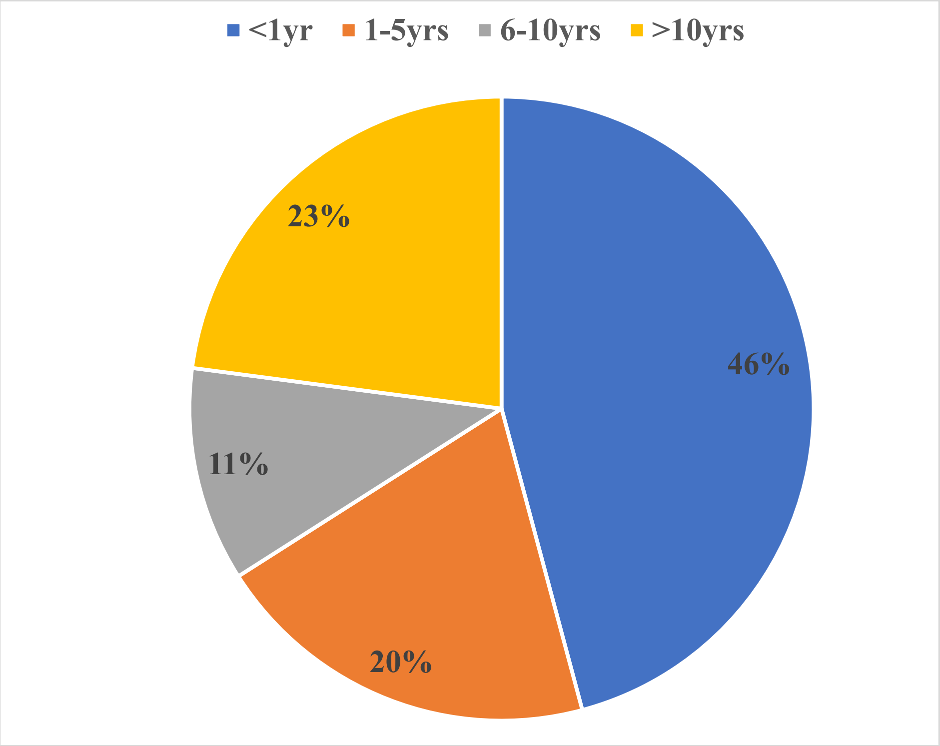


Table 3:- Distribution of patients according to gender

SEX	Frequency	Percent
Female	69	47.9
Male	75	52.1
Total	144	100.0

Majority were male patients (52.1%).

Figure 9:- Graph showing Distribution of subjects according to gender

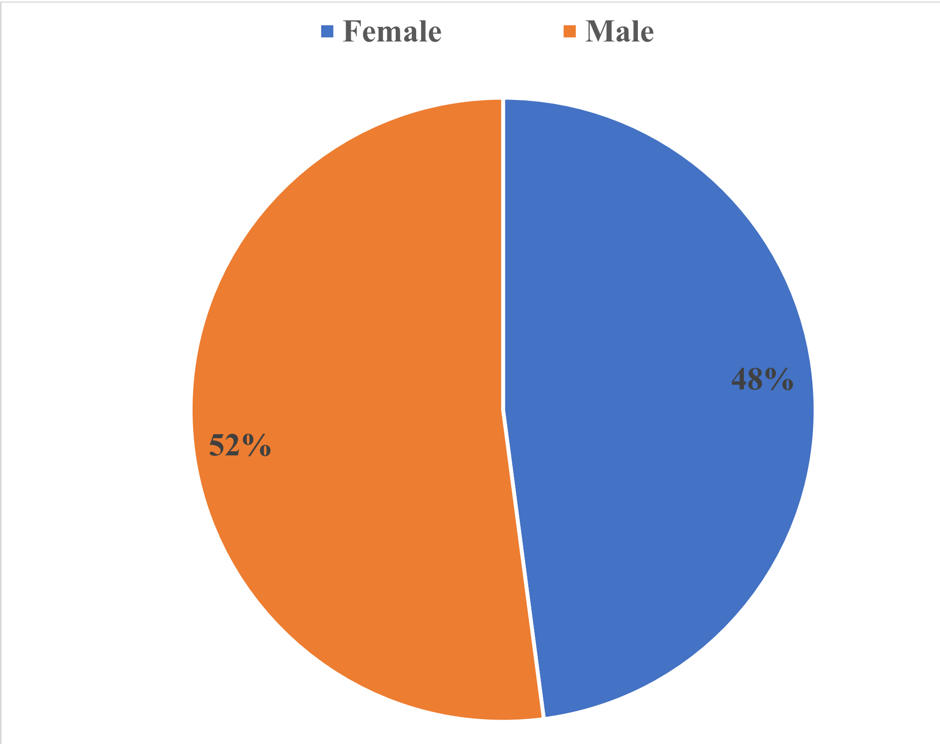


Table 4:- Distribution of patients according to Diagnosis

Diagnosis	Frequency	Percent
Acute Encephalitis Syndrome	1	.7
Acute GE with severe dehydration	31	21.5
Dengue Fever with warning signs	19	13.2
Diabetes Ketoacidosis	14	9.7
Drowning	2	1.4
OP Compound Poisoning	1	.7
Pubertal Menorrhagia	1	.7
Rat Poisoning with shock	1	.7
Respiratory Failure	1	.7
Severe Dengue Fever	12	8.3
Severe Sepsis in Shock	61	42.3
Total	144	100.0

- The most common etiology in children who presented with shock in our study was Sepsis in septic shock.Bronchopneumonia was the most common etiology of septic shock . 42% of the studied had septic shock. Most children presented with warm shock and hyperdynamic circulation . However few children presented with cold shock. Of the total 42 cases blood culture and sensitivity revealed growth of organism in 28 cultures. The organism which were isolated include Enterococcus, Klebsiella Pneumonia, Staphylococcus aureus, Acinetobacter species and Pseudomonas aeruginosa. The other cases had positive septic screen.

- The second most common etiology for shock was acute gastroenteritis with severe dehydration.31 % of the children studied had AGE with severe dehydration.
- 19(13.2%) children presented with Dengue fever with warning signs and 12 children had severe dengue fever(8.3%). Children with Dengue fever with warning signs presented with hypotension(compensated shock), thrombocytopenia, excessive vomiting, pain abdomen. These children were in the compensated phase and responded to fluid resuscitation. Children with Severe dengue fever presented with acute respiratory distress syndrome, Dengue hepatitis, Dengue hemorrhagic fever. Dengue encephalitis and Multi Organ dysfunction syndrome.
- 14(9.7%) children presented with Diabetic Ketoacidosis, Out of which 10 cases were newly diagnosed type I diabetes. And 4 cases were old cases of Type 1 diabetes with poor compliance to insulin therapy.All 14 children presented with severe Diabetic ketoacidosis.
- The various other etiologies for shock include, OP compound Poisoning, Drowning, late presentation of consumption of rat poison (Zinc Phosphide), and pubertal menorrhagia.

Figure 10:- Graph showing Distribution of patients according to Diagnosis

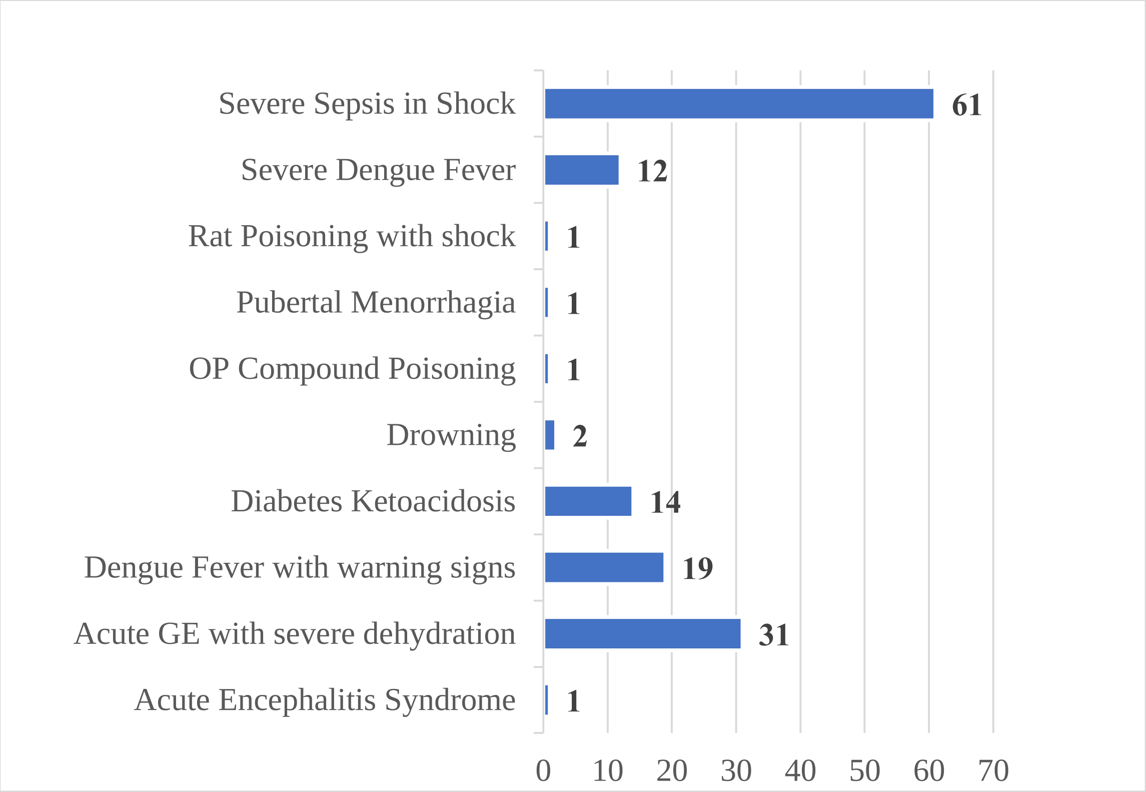


Table 5:- Distribution of patients according to age group and outcome

Age group	Outcome		Total
	Survivors	Non Survivors	
<1yrs	37	29	66
	56.1%	43.9%	100.0%
1-5yrs	24	5	29
	82.8%	17.2%	100.0%
6-10yrs	13	3	16
	81.3%	18.8%	100.0%
>10yrs	27	6	33
	81.8%	18.2%	100.0%
Total	101	43	144
	70.1%	29.9%	100.0%

P value 0.009, there was statistical significant difference found between age group and outcome

The mortality rate was higher in children less than 1 year of age.

Figure 11:- Graph showing distribution of patients according to age group and outcome

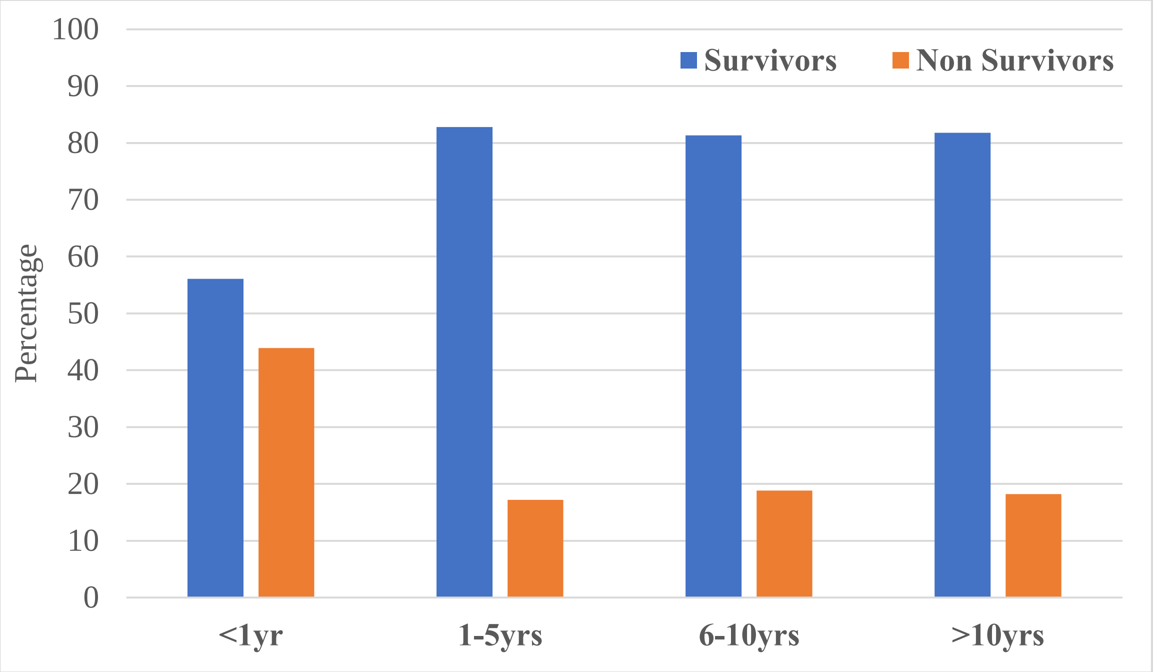
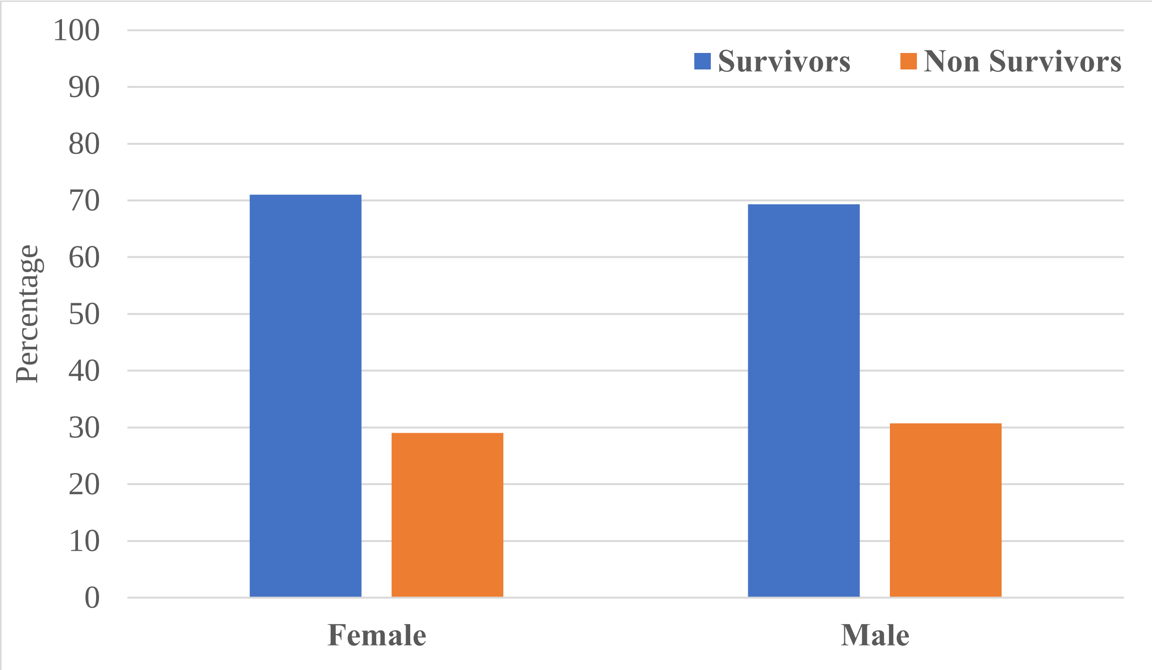


Table 6:- Distribution of patients according to gender and outcome

Sex	Out Come		Total
	Survivors	Non Survivors	
Female	49	20	69
	71.0%	29.0%	100.0%
Male	52	23	75
	69.3%	30.7%	100.0%
Total	101	43	144
	70.1%	29.9%	100.0%

P value 0.827 there was no statistical significant difference found between sex and outcome

Figure 12:- Graph showing distribution of patients according to gender and outcome



The various important biochemical parameters were Compared in survivors and non survivors(Arterial pH, Bicarbonate, Serum Potassium values, Blood Urea Nitrogen and serum creatinine)

Table 7: Comparison of arterial pH among survivors and non survivors at various interval

	Out Come	Mean	Std. Deviation	P value
0hrs	Survivors	7.05	0.14	<0.001
	Non survivors	6.88	0.14	
12hrs	Survivors	7.17	0.14	<0.001
	Non survivors	6.90	0.11	
24hrs	Survivors	7.28	0.12	<0.001
	Non survivors	6.97	0.18	

There was a statistically significant difference found between survivors and Non survivors with respect to arterial pH at all the time intervals

Figure 13:- Graph showing Comparison of arterial pH among survivors and non survivors at various interval

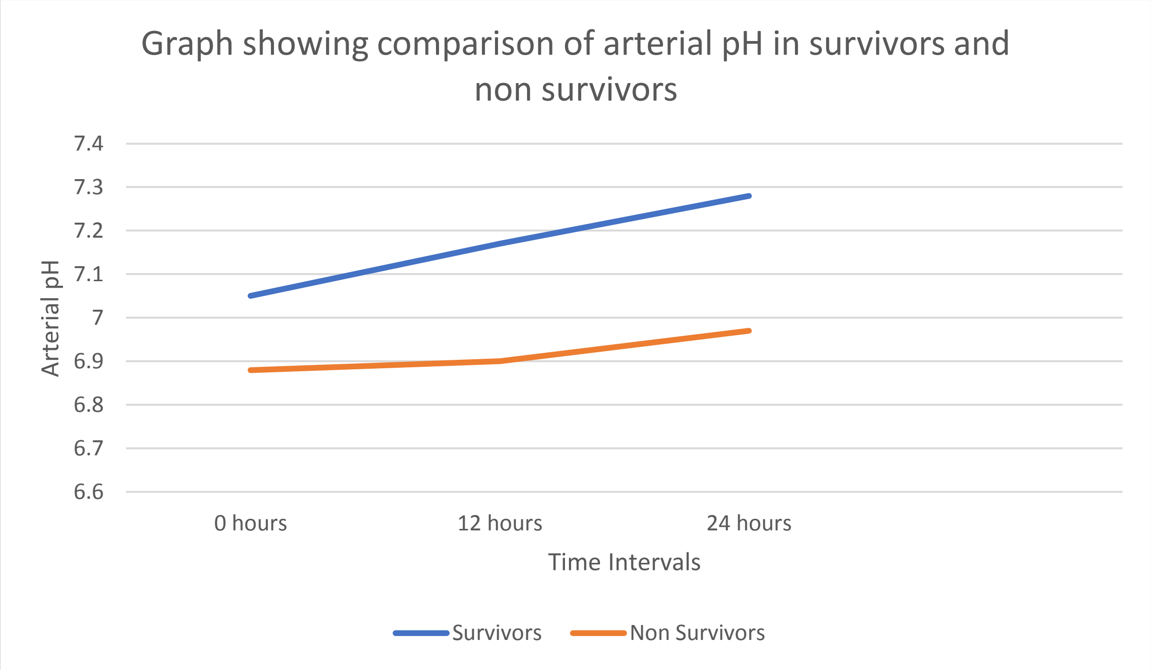


Table 8 :- Comparison of arterial bicarbonate among survivors and non survivors at various time intervals

	Out Come	Mean	Std. Deviation	P value
0hrs	Survivors	13.8	4.3	<0.001
	Non survivors	10.4	2.2	
12hrs	Survivors	16.4	4.1	<0.001
	Non survivors	10.7	2.4	
24hrs	Survivors	19.3	3.9	<0.001
	Non survivors	11.9	2.9	

There was a statistically significant difference found between survivors and Non survivors with respect to respect to arterial bicarbonate at all the time intervals

Figure 14:- Graph showing Serum Bicarbonate values among survivors and non survivors

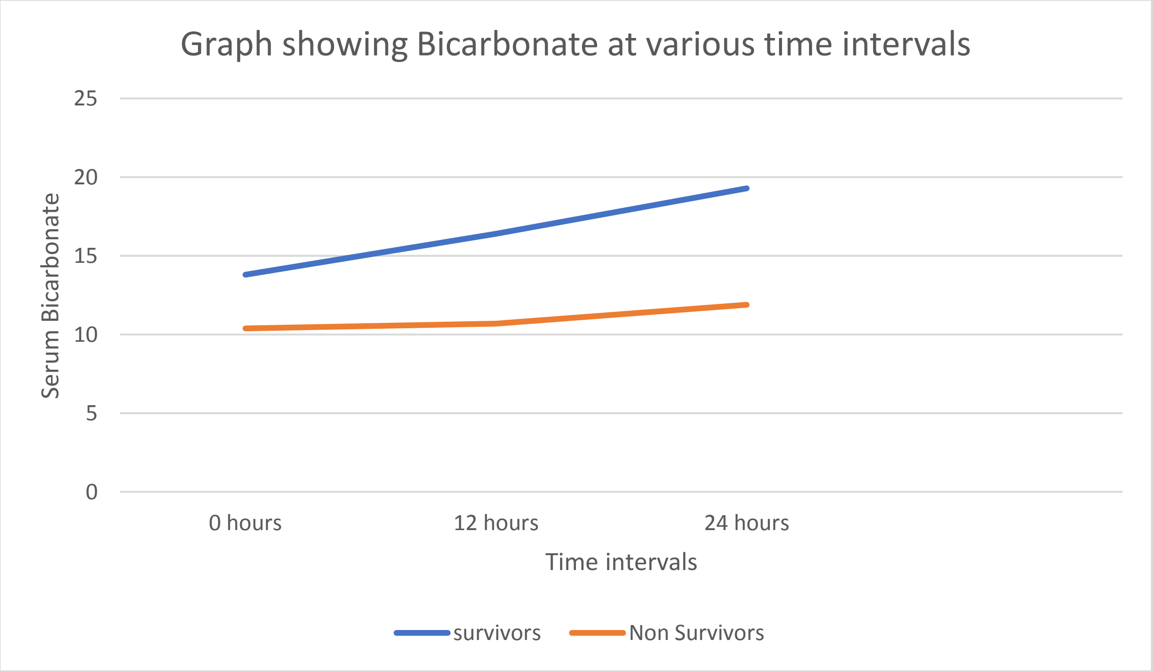


Table 9 :- Comparison of serum potassium values among survivors and non survivors at various time intervals

	Out Come	Mean	Std. Deviation	P value
0hrs	Survivors	4.8	0.9084	<0.001
	Non survivors	5.4	0.9218	
12hrs	Survivors	4.6	0.7419	<0.001
	Non survivors	5.54	0.898	
24hrs	Survivors	4.5	0.615	<0.001
	Non survivors	5.55	0.8123	

There was a statistically significant difference found between survivors and Non survivors with respect to serum potassium at all the time intervals.

Figure 15 :- Graph showing serum potassium values at various time intervals

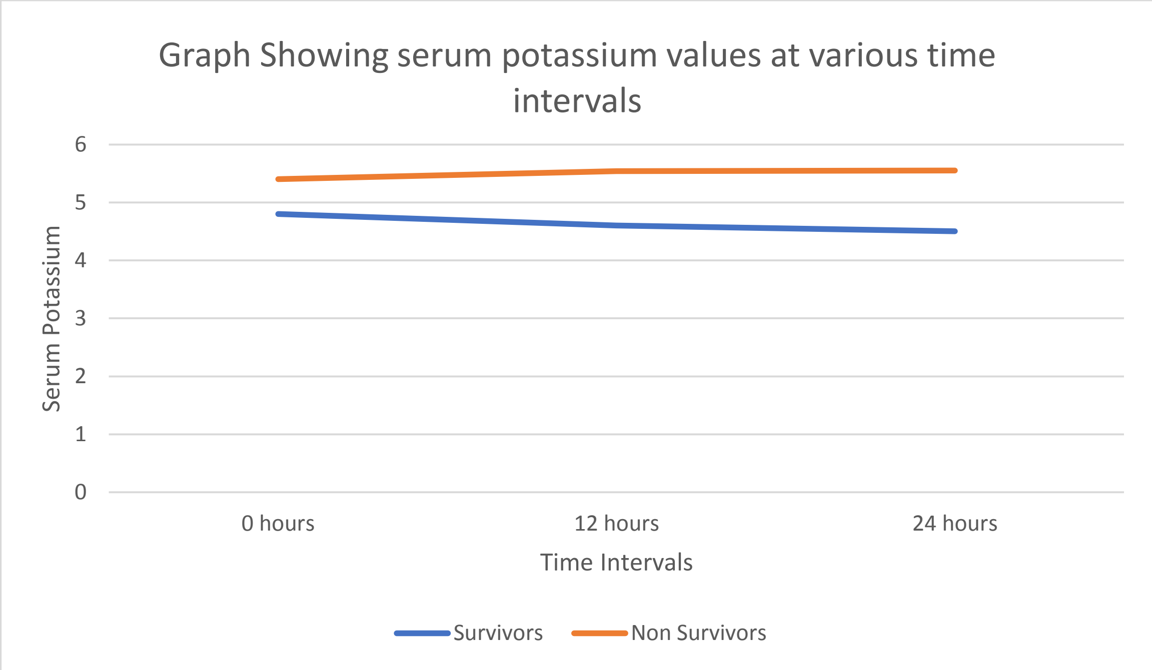


Table 10: Comparison of Blood Urea Nitrogen values among survivors and non survivors at various time intervals:

	Out Come	Mean	Std. Deviation	P value
0hrs	Survivors	23.2	14.0	<0.001
	Non survivors	40.6	18.3	
12hrs	Survivors	21.0	12.1	<0.001
	Non survivors	41.7	17.4	
24hrs	Survivors	19	13.0	<0.001
	Non survivors	39.0	19.0	

There was a statistically significant difference found between survivors and Non survivors with respect to respect to Blood urea nitrogen at all the time intervals

Figure 16:- Graph showing Blood urea nitrogen values at various time intervals

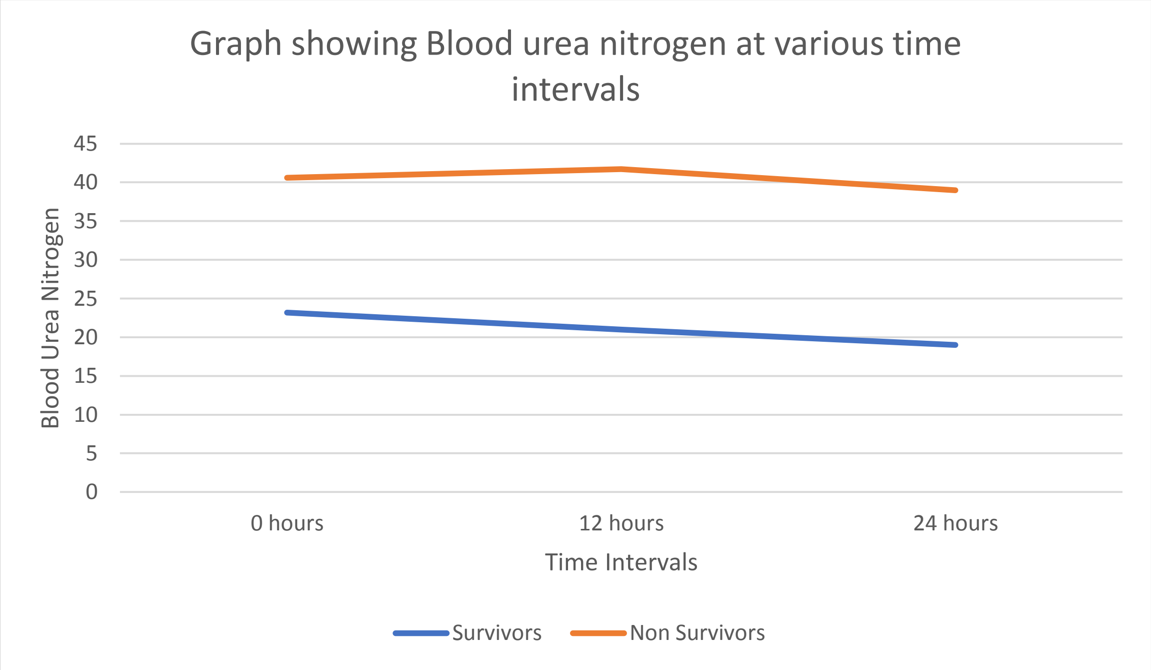


Table:- 11 Comparison of Serum creatinine values among the survivors and non survivors at various time intervals

	Out Come	Mean	Std. Deviation	P value
0hrs	Survivors	1.0	0.482	<0.001
	Non survivors	1.7	0.611	
12hrs	Survivors	0.90	0.382	<0.001
	Non survivors	1.6	0.611	
24hrs	Survivors	0.72	0.365	<0.001
	Non survivors	1.5	0.5719	

There was a statistically significant difference found between survivors and Non survivors with respect to respect to Serum Creatinine at all the time intervals

Figure 17:- Graph showing Serum creatinine values at various time intervals

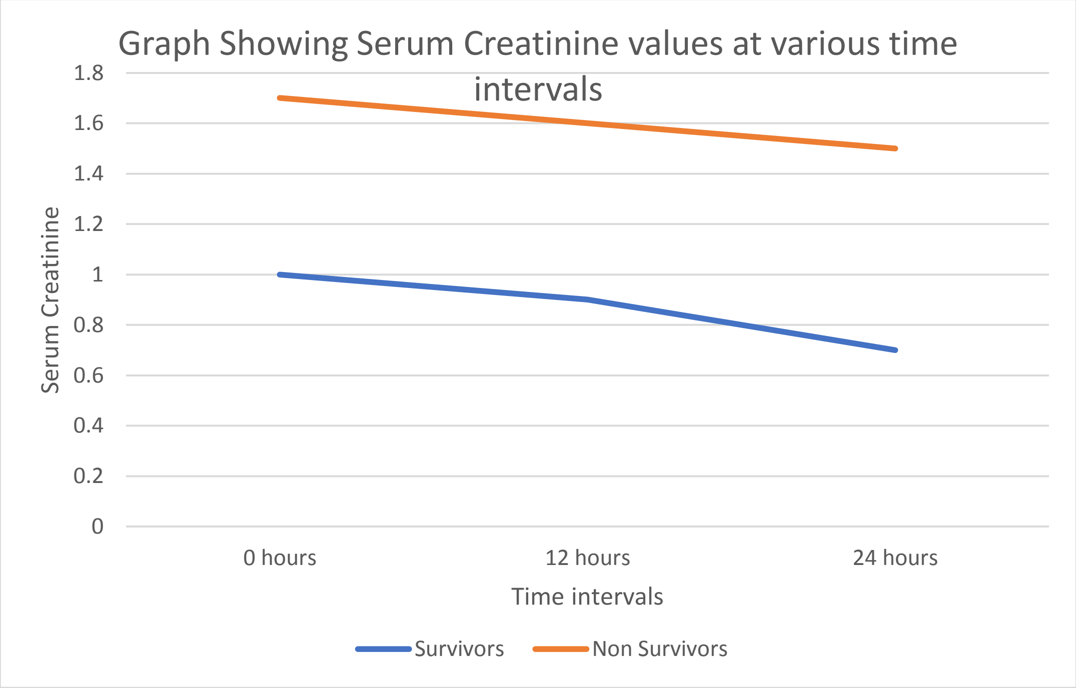


Table 12:- Comparison of serum lactate level among survivors and non survivors at various interval

	Out Come	Mean	Std. Deviation	P value
0hrs	Survivors	2.922	0.662	<0.001
	Non survivors	4.017	0.291	
12hrs	Survivors	2.488	0.627	<0.001
	Non survivors	4.250	0.319	
24hrs	Survivors	2.065	0.664	<0.001
	Non survivors	4.187	0.506	

- Mean serum lactate value measured at 0 hours, 12 hours and 24 hours of admission in survivors was found to be 2.92 mmol/l, 2.4mmol/l and 2.06 mmol/l respectively. The mean serum lactate values measured in non survivors at 0 hours , 12 hours and 24 hours of admission was 4.01mmol/l, 4.25mmol/l and 4.18mmol/l respectively.

- As it can be observed the serum lactate values in survivors at time of admission was very much lower in comparison to non survivors at the time of presentation. There is progressive reduction in serum lactate values in survivors.
- Serum lactate values of non survivors at presentation was elevated significantly when compared to survivors . In non survivors there is persistent hyperlactatemia even following resuscitation. Thus concluding that high serum lactate values at presentation (according to our study lactate values above 4mmol/l)and persistent hyperlactatemia are associated with increased mortality.
- There was a statistically significant difference found between survivors and Non survivors with respect to serum lactate levels at all the time intervals

Figure 18:- Graph showing Comparison of serum lactate level among survivors and non survivors at various interval

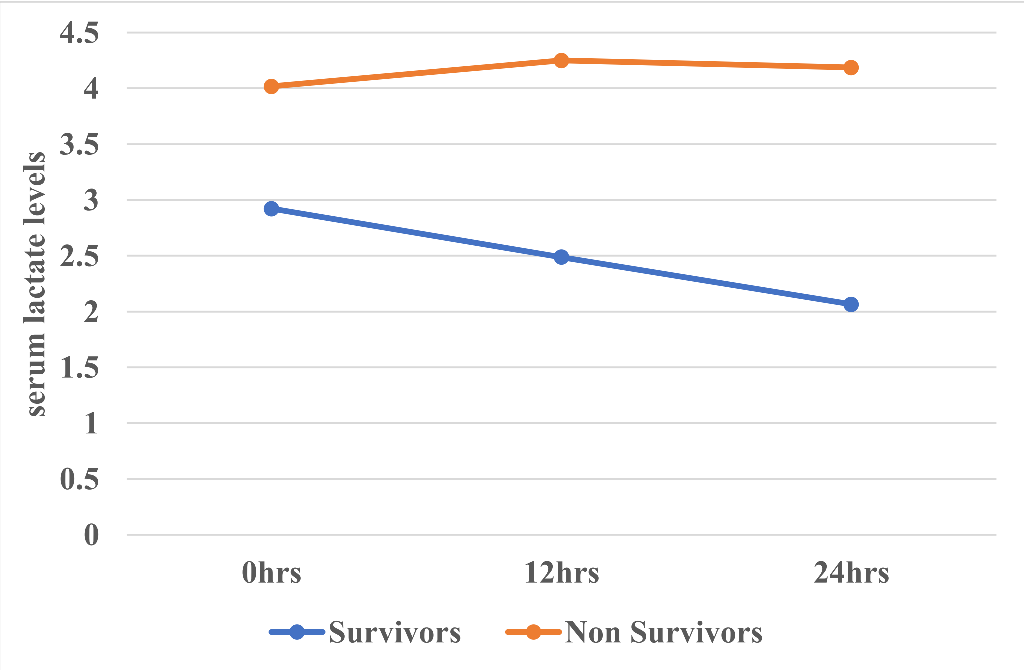


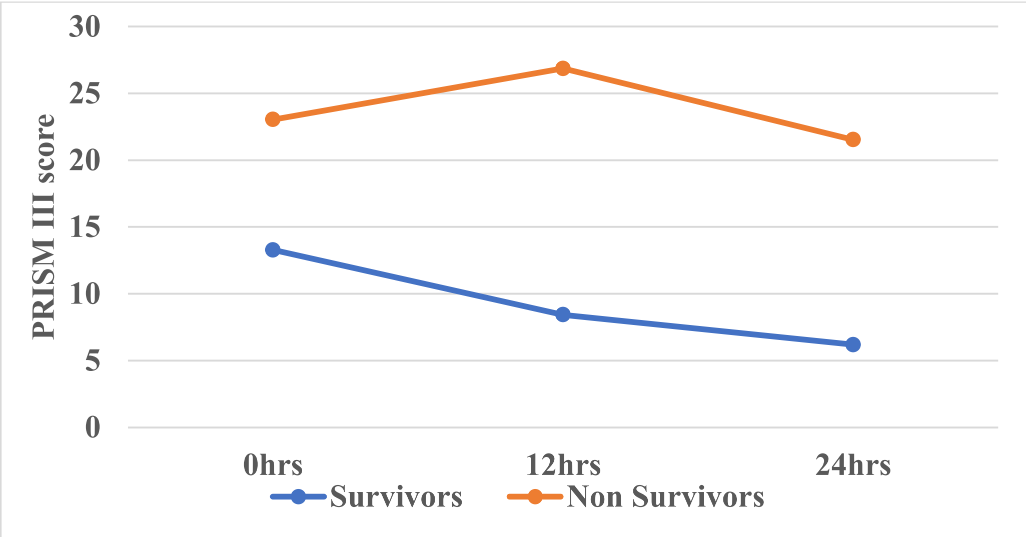
Table 13:- Comparison of PRISM III score among survivors and non survivors at various interval

	Out Come	Mean	Std. Deviation	P value
0hrs	Survivors	13.28	4.693	<0.001
	Non survivors	23.05	7.730	
12hrs	Survivors	8.43	3.371	<0.001
	Non survivors	26.86	9.486	
24hrs	Survivors	6.19	3.280	<0.001
	Non survivors	21.53	9.007	

- The mean PRISM III values measured at 0 hours, 12 hours and 24 hours of admission in survivors was found to be 13, 8 and 6 respectively. The mean PRISM III values measured in non survivors at 0 hours , 12 hours and 24 hours of admission was 23, 26 and 21respectively.
- The PRISM III values measured at admission in survivors was low and there was progressive reduction in the PRISM III scores.
- The PRISM III scores in non survivors was found to be elevated at presentation when compared to non survivors and there is increase in the scores at 12 hours of admission in non survivors.
- A persistently elevated scores above 20 was found to be associated with increase in the risk of mortality among children with shock

There was a statistically significant difference found between survivors and Non survivors with respect to PRISM III score at all the time intervals

Figure 19:- Graph showing Comparison of PRISM III score among survivors and non survivors at various interval



A highly significant positive correlation existed between the PRISM III score and lactate level at admission (r = 0.678; P = <0.001).

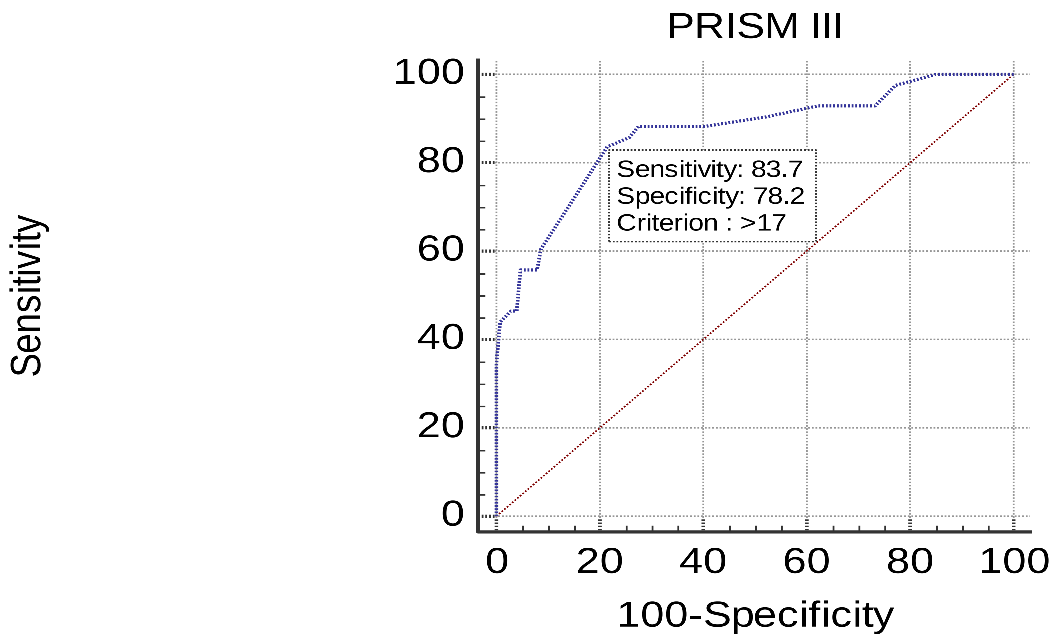
Table 14:- Comparison of area under the ROC curve for PRISM score and Serum lactate level

	PRISM III score	Serum lactate level
Area under the ROC curve (AUC)	0.866	0.958
Standard Error	0.0357	0.0171
95% Confidence interval	0.799 to 0.917	0.911 to 0.984
Significance level (P Value)	<0.0001	<0.0001

The area under the ROC curve for the serum lactate levels (0.958) suggests that it was a strong predictor of mortality in study subjects when compared to PRISM III score which had area under the ROC curve 0.866.

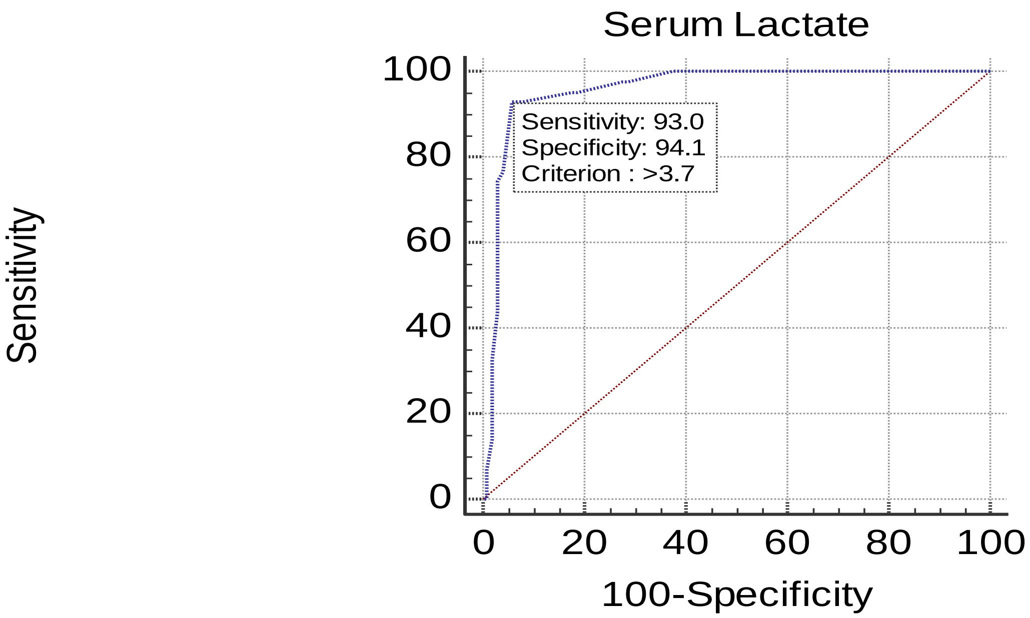
Area under the ROC curve for Both PRISM III score and serum lactate levels had a significant P value <0.001)

Figure 20:- Receiver operating characteristic ROC curves for PRISM III score



This is the ROC curve for the PRISM III scores. The x axis depicts specificity(true negative rate) rate and the y axis depicts the sensitivity (true positive rate). From this ROC curve it can be seen that the sensitivity of the PRISM III scores , that is the ability of the PRISM III scores to truly identify the children in shock who have poor prognosis and higher risk of mortality above the value of 17 is 83.7% and the specificity of PRISM III scores to identify the children who are not at the risk of mortality is 78.2 %.

Figure 21:- Receiver operating characteristic ROC curves for serum lactate



This is the ROC curve for the serum lactate values. The x axis depicts specificity(true negative rate) rate and the y axis depicts the sensitivity (true positive rate). From this curve it can be seen that sensitivity of the serum lactate values , that is ability of the serum lactate values to truly identify the children in shock who have poor prognosis and higher risk of mortality above the value of 3.7 is 93 and specificity of serum lactate values to identify the children who are not at risk of mortality is 94.1.

DISCUSSION

DISCUSSION

A total of 144 children were included in our study.Most of the children 66, (45 %) were in the age group of less than 1 year.29 children were in the age group of 1-5 years(20%). 16 children were between the age group of 6-10 years(11.1%) and 33 children were in the age group of >10 years(22.9%) 69 children were females (47.9%) and 75 children were males(52.1%).

It can be seen from fig.10 the most common etiology in children who presented with shock in our study was Sepsis in septic shock. Bronchopneumonia was the most common etiology of septic shock . **42%** of the studied had septic shock. Most children presented with warm shock and hyperdynamic circulation . However few children presented with cold shock. Of the total 61 cases sent for culture, blood culture and sensitivity revealed growth of organism in 28 cultures. The organism which were isolated include Enterococcus, Klebsiella Pneumonia, Staphylococcus aureus, Acinetobacter species and Pseudomonas aeruginosa. The other cases had positive septic screen.

The second most common etiology for shock was acute gastroenteritis with severe dehydration. **31 %** of the children studied had AGE with severe dehydration.

19(13.2%) children presented with Dengue fever with warning signs and 12 children had severe dengue fever(8.3%). Children with Dengue fever with warning signs presented with hypotension (compensated shock), thrombocytopenia, excessive vomiting, pain abdomen. These children were in the compensated phase and responded to fluid resuscitation. Children with Severe dengue fever presented with acute respiratory distress syndrome, Dengue hepatitis, Dengue hemorrhagic fever. Dengue encephalitis and Multi Organ dysfunction syndrome.

14 (9%) children presented with Diabetic Ketoacidosis, Out of which 10 cases were newly diagnosed type I diabetes and 4 cases were old cases of Type 1 diabetes with poor compliance to insulin therapy. All 14 children presented with severe Diabetic ketoacidosis. The various other etiologies for shock include, OP compound Poisoning, Drowning, late presentation of consumption of rat poison (Zinc Phosphide), and pubertal menorrhagia.

The important biochemical parameters that is arterial pH , bicarbonate levels, serum potassium levels, Blood urea nitrogen and serum creatinine were compared between survivors and non survivors.

Comparison of Arterial pH between survivors and non survivors

The mean arterial pH in survivors at 0 hours, 12 hours and 24 hours of admission was **7.05, 7.17 and 7.28**. And the mean arterial pH in non survivors at 0 hours , 12 hours and 24 hours of admission was **6.88, 6.9 and 6.97**. It was found that there was statistical significant difference(p value <0.001) in the arterial pH at various time interval between survivors and non survivors (fig 13). Thus ,persistent severe metabolic acidosis indicates poor prognosis in patients with shock.

Comparison of Bicarbonate between survivors and non survivors

Similarly bicarbonate values were compared in various time intervals in survivors and non survivors . The mean bicarbonate in survivors at 0 hours, 12 hours and 24 hours of admission was **13.2, 16.4 and 19.3**. And the mean bicarbonate in non survivors at 0 hours , 12 hours and 24 hours of admission was **10.4, 10.7and 11.9** It was found that survivors had a higher bicarbonate values at admission and various time intervals(fig.14).There was statistical significant (p value < 0.001) between survivors and non survivors with respect to bicarbonate values at various time intervals. Persistent low bicarbonate values in children presenting in shock is a poor prognostic indicator in patients in shock.

Correlation of Serum Potassium values in survivors and non survivors

Serum potassium values which are indicator of AKI which indirectly indicate pre renal failure in patients with shock was also compared in survivors and non survivors. The initial serum potassium levels in non survivors was elevated **(Mean : 5.4 mEq/l)** and a persistently elevated serum potassium levels above **5mEq/l** was seen in non survivors when compared to survivors(fig. 15). There was statistical significant difference(p value <0.001) in serum potassium levels in survivors and non survivors at various time intervals.

Comparison of Renal function between survivors and non survivors

Assessing renal function is a very good indicator of the circulatory function and it gives information of renal perfusion and status of intravascular fluid volume in patients in shock. In our study we compared the Blood urea nitrogen and serum creatinine values at various time intervals in survivors and non survivors. It was observed that non survivors had elevated blood urea nitrogen and serum creatinine when compared to non survivors at all the time intervals. Both these parameter were statistically significant between survivors and non survivors(p<0.001).The mean BUN values at various time interval in non survivors were **40.6mg/dl , 41.7 mg/dl and 39mg/dl**. All of which were persistently elevated(fig 16 and fig 17). Serum creatinine values in non survivors were persistently elevated in all the time intervals . The mean serum creatinine values at various time intervals in non survivors was **1.7mg/dl, 1.6 mg/dl and 1.5 mg/dl** at 0 hours, 12 hours and 24 hours following admission. Thus both the renal parameters considered had statistical significant difference in survivors and non survivors (p value <0.001).

Comparison of Blood lactate values between survivors and non survivors

Blood lactate levels in children who were admitted in shock was detectable between a range of **0.8 - 5.5mmol/l** in 144 samples collected. The mean serum lactate values measured at time of admission at 0 hours, 12 hours and 24 hours of admission in survivors was **2.92 mmol/l , 2.4mmol/l and 2.06 mmol/l** respectively. The mean serum lactate values measured in survivors at 0 hours, 12 hours and 24 hours of admission was **4.01 mmol/l , 4.25mmol/l and 4.18 mmol/l** respectively(fig 18).

BLOOD LACTATE VALUES AS A PROGNOSTIC MARKER IN PREDICTING MORTALITY

The ability of blood lactate values in predicting the mortality of all patients admitted with shock was assessed . Mean serum lactate value measured at 0 hours, 12 hours and 24 hours of admission in survivors was found to be 2.92 mmol/l, 2.4mmol/l and 2.06 mmol/l respectively. The mean serum lactate values measured in non survivors at

0 hours , 12 hours and 24 hours of admission was 4.01mmol/l, 4.25mmol/l and 4.18mmol/l respectively. As it can be observed, the serum lactate values in survivors at time of admission was very much lower in comparison to non survivors at the time of presentation. There is progressive reduction in serum lactate values in survivors. Serum lactate values of non survivors at presentation was elevated significantly when compared to survivors. In non survivors there is persistent hyperlactatemia even following resuscitation. Thus concluding that high serum lactate values at presentation (according to our study lactate values above 4mmol/l)and persistent hyperlactatemia are associated with increased mortality. There was a statistically significant difference found between survivors and Non survivors with respect to serum lactate levels at all the time intervals

It was observed that blood lactate values was found to be important useful tools in predicting mortality in patients in shock , the values achieved (fig.21) **AUC of 0.958 with standard error of 0.0171 with 95% confidence interval between 0.911 – 0.984**.

Many studies done previously have demonstrated that lactate values at admission or peak lactate values at any given time of admission is associated with mortality in adults. However a very few studies have been conducted in children and demonstrated the use of hyperlactatemia as a prognostic indicator of mortality in critically ill children in PICU.

It has been observed in our study that severity and extent of hyperlactatemia is a strong independent predictor of mortality in critically ill children presenting with shock.

Correlation of serum lactate values and PRISM III scores in survivors and non survivors

In our study we have correlated serum lactate values to PRISM III scores and compared as to which is a better indicator in predicting mortality in children presenting with shock. PRISM III score is a good valid measure of illness severity of the critically ill children during the initial 24 hours of admission. It reflects on overall clinical picture of the child. It is the sum total of the physiological, biochemical and hematological parameters assessed. The mean PRISM III values measured at 0 hours, 12 hours and 24 hours of admission in survivors was found to be **13, 8 and 6** respectively. The mean PRISM III values measured in non survivors at 0 hours , 12 hours and 24 hours of admission was **23, 26 and 21** respectively. The PRISM III values measured at admission in survivors was low and there was progressive reduction in the PRISM III scores.The PRISM III scores in non survivors was found to be elevated at presentation when compared to non survivors and there is increase in the scores at 12 hours of admission in non survivors. A persistently elevated scores above 20 was found to be associated with increase in the risk of mortality among children with shock.

The AUC (fig. 20) for PRISM III scores was 0.866 with standard error of 0.0357 wit 95% confidence interval of 0.799 to 0.917.

There was a statistically significant difference found between survivors and Non survivors with respect to PRISM III score at all the time intervals **Area under ROC curve was compared between serum lactate values and PRISM III scores. Area under ROC curve for serum lactate values was found to be 0.958 and area under**

ROC curve for PRISM III score was 0.866. Hence this proves that serum lactate values are better predictors of mortality when compared to PRISM III score.

Area under ROC curve for PRISM III score and serum lactate levels had a significant P value <0.001.

In our study sensitivity and specificity of serum lactate values and PRISM III scores was determined along with positive predictive values and negative predictive values. Using this data ROC curve was plotted. Also, optimal cut off values were obtained for determining mortality.

Table 14:- Cut-off points for the PRISM III score and lactate levels along with Sensitivity, specificity, PPV and NPV of PRISM III score and serum lactate levels

	Sensitivity	Specificity	PPV	NPV
PRISM III score (>15)	88.37	72.28	57.6	93.6
PRISM III score (>17)	83.72	78.22	62.1	91.9
Serum lactate (>3.5)	95.35	82.18	69.5	97.6
Serum lactate (>3.7)	93.0	94.1	87	96.9

75% of the children included in our study had a blood lactate concentration of >2.5mmol/l at presentation. Blood lactate values had a sensitivity of 95.5 and specificity of 82.18% at values greater than 3.5mmol/l with a PPV of 69.5 and NPV of 97.6 and at values >3.7 blood lactate values had a sensitivity of 93% and specificity of 94.5% with a PPV of 87 and NPV of 96.9.

Overall any blood lactate values above 4mmol/l within initial 24 hours of admission even after extensive resuscitation indicated very poor prognosis and higher risk of in hospital mortality.

The above analysis was compared with PRISM III scores which at values greater than 15 had a sensitivity of 88.3 and a specificity of 72.28 with PPV of 57.6 and NPV of 93.6 and at values greater than 17 it displayed a sensitivity of 95.35 and a specificity of 78.22 with a PPV of 62.1 and a NPV of 91.9. And it was observed that a PRISM III score of greater than 20 at any given time of admission within initial 24 hours indicates poor prognosis and carries a higher risk of mortality.

Our study was compared to a study done by **Bai Z et al**[2], in assessing blood lactate levels as prognostic indicator in critically ill children admitted to PICU. Whereas our study included children who were presenting with shock, this study included all the critically ill children presenting to PICU. A total of 1109 children were included. Median blood lactate levels were found to be 3.2 mmol/l(2.2-4.8mmol/l). This study showed that elevated blood lactate levels at admission was associated with greater risk of mortality (odds ratio [OR] = 1.38; 95% confidence interval [CI], 1.30-1.46; p <0.001). It was demonstrated that elevated blood lactate level, and a high PRISM III scores were independent risk factors for mortality in all critically ill children. In their study blood lactate had an AUC of 0.79 (p <0.001) for predicting mortality that was similar to that of PRISM III AUC = 0.82;(p <0.001). However as discussed above in our study it was observed that Blood lactate levels were single important parameter which is better than PRISM III scores in predicting mortality in children presenting with shock[2].

While our study correlated PRISM III scores and Serum lactate values, **Morris**[5] others conducted a retrospective cohort study in PICU to investigate whether blood lactate concentration on admission predicts mortality in paediatric intensive care and if its addition can improve the performance of the Paediatric Index of Mortality 2 (PIM2) mortality prediction score and also to compare as to which is a better predictor of mortality. Both PRISM III and PIM score are used to predict mortality. While our study included only 144 children in shock, This study included total of 2,380 children All children irrespective of etiology admitted to PICU were include in this study.. Total number of non survivors was 155. It was observed that the admission lactate in non-survivors was higher than in survivors (mean [standard deviation, SD]) 6.6 [5.6] versus 3.0 [2.5] mmol/l, had a positive association with mortality [adjusted odds ratio (OR) for death per unit (mmol/l)] increase 1.11 [95 % confidence interval (CI) 1.06–1.16; $p < 0.001$] and significantly improved the model fit of PIM2 when it replaced absolute base excess. Thus this study concluded that PICU admission blood lactate concentration predicts mortality independently of PIM2 [5].

Another similar study was conducted by **K R Jat**[29] et al. This study was conducted to assess serum lactate values as prognostic indicator of mortality in children admitted to PICU with septic shock whereas in our study we studied patients presenting with all etiologies of shock. Out of the total 250 children admitted in PICU during the study period 46 patients were diagnosed with septic shock and were included in the study. Similar to our study pneumonia was the most common cause of septic shock in this study. Serum lactate values and PRISM III scores were recorded at 0 hours, 12 hours and 24 hours of admission.It was observed that the initial as well as the subsequent lactate values were higher in the non survivors when compared to survivors.In our study it was observed that serum lactate values greater than 4mmol/l at any given time interval from admission to 24 hours indicated poor prognosis whereas in their study it was observed that a serum lactate value of more than 5mmol/l during the initial 24 hours of resuscitation was good predictor of mortality. They also correlated serum lactate values with PRISM III scores and observed that highly positive correlation existed between serum lactate and PRISM III scores. A PRISM III score greater than 10 and a lactate values greater than 5mmol/l at all time periods discriminated survivors from non survivors [29].

Kim Y A[1] and others conducted a retrospective study on paediatric patients presenting with septic shock at Seoul Korea.This study also included only children presenting with septic shock. 65 patients were enrolled and overall mortality of these patients was studied.In our study we assessed blood lactate values at 0 hours, 12 hours and 24 hours whereas in this study serial blood lactate levels was assessed at the time of admission and every 6 hourly after admission upto 24 hours. We assessed only blood lactate values whereas in this not only assessed lactate values but also other lactate associated parameters that is lactate clearance and also lactate areas were also studied as predictors of mortality Survivors, in comparison with non survivors had an initial lactate values of 3.13+/- 2.79 vs 6.16 +/- 4.87 mmol/l ; a lactate clearance of 32.8+/- 63.4 vs -30.8+/- 75.6% and lactate area of 59.7+/- 56.0 vs 168+/- 107 mmol/l. Hence this study concluded that blood lactate values and also lactate associated parameters were potentially very useful markers of mortality[1]

Chaudhary J[3] and others conducted prospective observational study including only septic shock patients between age group of 1month to 12 years. Our study included patients presenting with all types of shock and included children between age group of 1month and 18 years. Whereas in our study we observed that serum lactate values greater than 4 mmol/l at any given time during the first 24 hours of admission, in this study it was observed that the serum lactate values greater than 5 mmol/l in children presenting with septic shock indicates poor prognosis and is a good predictor of mortality[1].

Thus as it can be observed from our study as well as previous other similar studies that elevated serum lactate is a single useful reliable parameter which can be used as a prognostic indicator for mortality in patients presenting with shock. PRISM III scores gives a overall clinical picture of the child and can be used for monitoring of critically ill children admitted to PICU. Both Serum lactate and PRISM III are useful predictors of mortality in children in Shock admitted to PICU.

CONCLUSION

CONCLUSION

- Majority of the children who presented in shock were below the age group of 1 year.
- Severe sepsis in septic shock was the most common etiology of shock in children.
- Bronchopneumonia was the most common cause of septic shock in children.
- Out of the total blood culture samples sent only 45 % cases were culture positive. Other cases were septic screen positive.
- Acute gastroenteritis with severe dehydration was the second most common cause of shock in the studied subjects.
- Important biochemical parameters (arterial pH, Bicarbonate, Serum Potassium, BUN and Serum Creatinine) were studied between survivors and non survivors.
- It was observed that all of these biochemical parameters had a statistical significant difference (p value < 0.001) between survivors and non survivors. Hence all these parameters can be used as prognostic indicators and predictors of mortality in children presenting with shock.
- Serum lactate were assessed between survivors and non survivors at the time of admission 0 hours, 12 hours and 24 hours.
- It was observed that there was persistent hyperlactemia in non survivors and serum lactate values persistently greater than 4mmol/l within the first 24 hours of admission were associated with greater chances of mortality.

- The PRISM III scores were recorded using the various physiological, biochemical and hematological parameters. The sum of the PRISM III scores were compared in between survivors and non survivors.
- It was observed that the non survivors had persistently elevated PRISM III scores when compared to non survivors. Whereas survivors not only had low PRISM III scores initially and later showed a reducing trend.
- Both PRISM III scores and serum lactate values are good predictors of mortality . However serum lactate values were better compared PRISM III scores in predicting mortality.
- Serum lactate value is a single independent useful data that can be used as a predictor of mortality which is better and more feasible than PRISM III scores which uses many variables and multiple physiological, biochemical and hematological parameters.

SUMMARY

SUMMARY

- This study was a prospective observational study conducted in tertiary care centre at RL Jalappa Hospital Kolar.
- The aim of this study was to evaluate serum lactate levels as a prognostic indicator in children presenting with shock to our hospital.
- And to correlate the serum lactate values obtained at various time intervals (0 hours, 12 hours and 24 hours) with PRISM III scores calculated as a sum total of various physiological parameters, biochemical parameters and hematological parameters recorded at the same time intervals.
- All the children presenting with shock to Pediatrics intensive care unit, irrespective of the etiology and the stage of shock were included in our study.
- Exclusion criteria included children who were discharged against medical advice and died within 2 hours of admission.
- A total of 144 children were included in our study. Majority of the children were of age group less than 1 year of age. 45 % of the children belonged to age group less than 1 year.
- All the children were assessed with thorough, clinical examination and relevant investigations.
- The most common etiology of shock in our study was found to be Septic shock (43%)
- The most common etiology for septic shock was Bronchopneumonia and the second most common etiology of shock was acute gastroenteritis with severe dehydration.

- The other etiologies of shock included Diabetic Ketoacidosis, Near Drowning, late presentation of rat poison consumption, Dengue fever(Dengue fever with warning signs and severe dengue fever) and Pubertal menorrhagia
- Important biochemical parameters, serum lactate values and PRISM III values were compared between the survivors and non survivors.
- Among the important Biochemical parameters compared (arterial pH, Bicrbonate, serum potassium, BUN and serum creatinine) there was statistical significant difference between survivors and non survivors (p value < 0.001) at all time intervals. Thus all these parameters are very important prognostic marker of mortality.
- Blood lactate levels in children with shock was detectable with a range of 0.8 - 5.5mmol/l in 144 samples collected There was statistical significant difference(p value < 0.001) between survivors and non survivors in serum lactate values assessed at all the time intervals. The mean serum lactate values measured at the time of admission at 0 hours, 12 hours and 24 hours of admission in survivors was 2.92 mmol/l , 2.4mmol/l and 2.06 mmol/l respectively. The mean serum lactate values measured in survivors at 0 hours, 12 hours and 24 hours of admission was 4.01 mmol/l , 4.25mmol/l and 4.18 mmol/l respectively. In non survivors serum lactate values were found to be persistently elevated in all the time intervals. There was persistent hyperlactatemia of mean serum lactate values of >4mmol/l in non survivors in all the time intervals. The area under the ROC curve for the serum lactate levels (0.958) suggests that it was a strong predictor of mortality in study subjects.

- PRISM III scores were compared between the study subjects at various time intervals. The mean PRISM III values measured at 0 hours, 12 hours and 24 hours of admission in survivors was found to be **13, 8 and 6** respectively. The mean PRISM III values measured in non survivors at 0 hours, 12 hours and 24 hours of admission was **23, 26 and 21** respectively. The PRISM III values measured at admission in survivors was low and there was progressive reduction in the PRISM III scores.The PRISM III scores in non survivors was found to be elevated at presentation when compared to non survivors and there is increase in the scores at 12 hours of admission in non survivors. A persistently elevated scores above 20 was found to be associated with increase in the risk of mortality among children with shock.
- The area under the ROC curve for PRISM III score (0.866) suggests that it was also a good predictor of mortality.
- The area under the ROC curve for the serum lactate levels (0.958) suggests that it was a strong predictor of mortality in study subjects when compared to PRISM III score which had area under the ROC curve 0.866.
- Area under the ROC curve for Both PRISM III score and serum lactate levels had a significant P value <0.001
- Thus it can be concluded from our study that serum lactate values are single most important independent prognostic indicators of mortality when compared with PRISM III scores.

RECOMMENDATIONS OF THE STUDY

- Serum lactate value is an important prognostic marker in children in shock.
- It should be routinely used in PICU for monitoring of the patients in shock and early intervention can be followed.
- Serum Lactate along with PRISM III scores gives an overall picture of the critically ill children and can be used in assessing the clinical status of the patients in PICU.

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BIBILIOGRAPHY

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ANNEXURES

Prospective Study of Blood Lactate Levels as a Prognostic Indicator In patients with shock admitted to PICU in tertiary care centre

PROFORMA

Name:

Age:

Gender:

Address:

Chief Complaints:

History of Presenting Illness:

Past History:

Birth History:

Immunization History:

Developmental History:

General Physical Examination:

Vital Parameter

Parameters	0 hours	12 hours	24hours
Blood Pressure			
Heart Rate			
Temperature			
Respiratory Rate			
GCS			

Systemic Examination

Cardiovascular System:

Respiratory System:

Per abdomen examination:

Central Nervous System:

Investigations:

Arterial Blood Gas analysis:

Parameters	0 hours	12 hours	24hours
Arterial pH			
pCO2			
Total CO2			
PaO2			

Bicarbonate			

Biochemical Parameters:

Parameters	**0 hours**	**12 hours**	**24hours**
Blood Glucose			
Serum Potassium			
BUN			
Serum Creatinine			

Hematological Parameters:

Parameters	**0 hours**	**12 hours**	**24hours**
WBC Count			
Platelet Count			
PT			
APTT			

Serum Lactate Values : 0 hours:

12 hours:

24 hours:

PRISM III Scores: 0 hour:

12 hours:

24 hours:

INFORMED CONSENT

Name of the Study:Prospective Study of Blood Lactate Levels as a Prognostic Indicator In patients with shock admitted to PICU in tertiary care centre.

Chief researcher/ PG Guide's name: Dr.Krishnappa J

Principal investigator: Dr.Sri Raksha.S

Name of the subject: Age : Gender :

a. I have been informed in my own language that this study "Prospective Study of Blood Lactate Levels as a Prognostic Indicator In patients with shock admitted to PICU in tertiary care centre" involves the examination of my child along with arterial blood and venous blood sample will be drawn from my child for the purpose of evaluation and management of my child. I have been explained thoroughly and understand its complication and possible side effects.

b. I understand that the medical information produced by this study will become part of institutional record and will be kept confidential by the said institute.

c. I understand that my participation is voluntary and may refuse to participate or may withdraw my consent and discontinue participation at any time without prejudice to my present or future care at this institution.

d. I agree not to restrict the use of any data or results that arise from this study provided such a use is only for scientific purpose(s).

e. I confirm that ____________________ (chief researcher/ name of PG guide) has explained to me the purpose of research and the study procedure that I will undergo and the possible risks and discomforts that i may experience, in my own language. I hereby agree to give valid consent to participate as a subject in this research project.

Signature/ Thumb impression of the Parents/ Guardian:

Date:

Time:

I have explained to ___________________________ (subject) the purpose of the research, the possible risk and benefits to the best of my ability.

Chief Researcher/ Guide signature

Date:

Time:

Name of the Study:Prospective Study of Blood Lactate Levels as a Prognostic Indicator In patients with shock admitted to PICU in tertiary care centre.

PATIENT INFORMATION SHEET

Principal Investigator: Dr. Sri Raksha.S / Dr. Krishnappa J

I, Dr. Sri Raksha.S, post-graduate student in Department of Pediatrics at Sri

Devaraj Urs Medical College, will be conducting a study titled -Prospective Study of Blood Lactate Levels as a Prognostic Indicator In patients with shock admitted to PICU in tertiary care centre for my dissertation under the guidance of

Dr. Krishnappa J, Professor, Department of Pediatrics. Yours child will be subjected to thorough clinical examination and arterial and venous blood samples will be drawn for the evaluation and management of shock. Samples for serum lactate will be collected while collecting samples for ABG.

In this study, we will clinically assess your child and investigate for various blood parameters as a part of work up for management of shock .Blood samples will be drawn for assessing serum lactate values while collecting samples for ABG . Your child will be assessed for the various parameters at admission , after 12 hours of admission and after 24 hours of admission.

You will not be paid any financial compensation for participating in this research project. All of your child's data will be kept confidential and will be used only for research purpose by this institution. You are free to allow your child to be a part of the study. You can also withdraw your child from the study at any point of time without giving any reasons whatsoever. Your refusal to participate will not prejudice you to any present or future care at this institution.

Name and Signature of the Principal Investigator

Date

KEY TO MASTERCHART

BP: BLOOD PRESSURE

HR: HEART RATE

RR: RESPIRATORY RATE

PRISM III: PEDIATRIC RISK OF MORTALITY SCORE

PT : PROTHROMBIN TIME

PTT : PARTIAL THROMBOPLASTIN TIME

MASTER CHART

Name	Age	Sex	IP number	Diagnosis	BP	HR	Temperature	RR	Pupils	GCS	pH	pCO2	Total CO2	PaO2	Bicarbonate	Glucose	Potassium	BUN	Creatinine	WBC	Platelet	PT	PTT	Serum Lactate	PRISM III	Out Come
Kusuma	3 years	Female	548217	Acute GE with severe dehydration	60/40	130/min	96	42/min	Bilateral Equal and reactive	13/15	7.21	22	22	167	17.3	20	4.4	12	0.4	13k/cumm	530k/cumm	12.3	32.3	2.1mmol/l	5	Recovered
					66/52	110/min	98	36/min	Bilateral Equal and reactive	14/15	7.28	25	23.2	162	18.6	92	4.2	9	0.4	14k/cumm	500k/cumm	13.4	33.2	1.6mmol/l	3	
					76/80	100/min	98	32/min	Bilateral Equal and reactive	15/15	7.35	32	29.3	120	22	102	4	8	0.5	12k/cumm	450k/cumm	14.2	32.2	1.4mmol/l	3	
Izan Ahmed	2 month	Male	543926	Acute GE with severe dehydration	48/30	190/min	96	64/min	Bilateral Equal and reactive	GCS 11	7.1	20mmhg	17.8	120	13.2	38	4.2	15	0.4	14/cumm	549k/cumm	12.4	33.4	2.3mmol/l	5	Recovered
					58/40	176/min	98	48/min	Bilateral Equal and reactive	GCS 12	7.28	30	24.5	124	17.6	92	4.3	14	0.5	10k/cumm	455k/cumm	11.6	35.3	2mmol/l	5	
					68/40	154/min	99	40/min	Bilateral Equal and reactive	GCS 14	7.35	38	28.74	120	20.2	102	4	16	0.5	10k/cumm	400k/cumm	15.6	32.3	1.5mmol/l	5	
Hasini	3 months	Female	557721	Severe Sepsis in Shock	not recordable	198/min	96	78/min	Bilateral Equal and reactive	GCS 8/15	6.9	5.6	11.2	98	10	98	5.6	15	0.5	30k/cumm	815/cumm	23.2	40.4	3.9mmol/l	19	Death
					not recordable	176/min	98	44on mv	Bilateral pupils dilted and fixed	GCS 8/15	7	22	13.06	106	8	100	5	15	0.6	32k/cumm	615k/cumm	19.5	45.3	4.5mmol/l	21	
Nayara	2months	Female	584972	Bronchopneumonia , Sepsis in Shock	not recordable	182/min	100	62/min	Bilateral pupils dilted and fixed	GCS 12/15	7.16	17.8	16.2	128	12.3	132	6.9	10	0.5	25k/cumm	477k/cumm	13.4	33.4	2.8mmol/l	13	Recovered
					not recordable	140/min	98	66/min	Bilateral Equal and reactive	GCS 10/15	7.1	18.4	14.4	140	10.2	94	7.1	9	0.5	30k/cumm	400k/cumm	16.7	32.3	2.5mmol/l	13	
					48/38	178/min	98	40on mv	Bilateral Equal and reactive	GCS 8/15	6.9	20	13.2	80	8.6	102	5	16	0.9	34k/cumm	312k/cumm	13.7	30.5	3.1mmol/l	21	
Sowmya	5 years	Female	571899	Acute GE with severe dehydration	92/60	142/min	98	56/min	Bilateral Equal and reactive	GCS 15/15	7.2	30	26.9	130	20	131	4.6	10	0.4	10.5k/cumm	86k/cumm	21.2	43.3	1.7mmol/l	2	Recovered
					94/60	130/min	99	54/min	Bilateral Equal and reactive	GCS 15/15	7.3	34	28.2	126	21.2	120	4.2	9	0.4	7.35k/cumm	62k/cumm	13.4	36.3	1.5mmol/l	0	
					94/60	120/min	98.6	50/min	Bilateral Equal and reactive	GCS 15/15	7.34	38	29.8	120	22.6	98	4.2	12	0.5	12.67k/cumm	87k/cumm	14.5	33.2	1.1mmol/l	0	
Hemashree	10 years	Female	574123	Diabetes Ketoacidosis	90/60	132/min	99	44/min	Bilateral Equal and reactive	GCS 10/15	6.79	10.4	10.5	128	8.2	HIGH	5.1	15	0.5	33750/Cumm	522k/cumm	15.6	46.4	3.6mmol/l	11	Recovered
					94/50	120/min	99	36/min	Bilateral Equal and reactive	GCS 12/15	7.06	9.4	15.5	110	13.4	625	3.9	15	0.6	28k/cumm	450k/cumm	17.2	37.3	3mmol/l	7	
					98/60	100/min	99.6	30/min	Bilateral Equal and reactive	GCS 13/15	7.18	14.3	20.8	114	17.6	277	4.7	12	0.5	22k/cumm	450k/cumm	13.5	34.3	2.3mmol/l	7	
Keerthi	13 years	female	521489	Pubertal Menorrhagia	70/50	170/min	96	30/min	Bilateral Equal and reactive	GCS 13/15	7.35	32	25	128	18.2	110	4.3	10	0.4	9k/cumm	300k/cumm	11.3	33.2	1.1mmol/l	4	Recovered
					82/50	162/min	98	28/min	Bilateral Equal and reactive	GCS 15/15	7.38	34	27	120	20.3	120	4.2	9	0.5	9.2k/cumm	320k/cumm	17.8	32.5	1mmol/l	0	
					90/60	140/min	98	24/min	Bilateral Equal and reactive	GCS 15/15	7.4	35	30.45	110	22.4	110	4.2	9	0.4	10k/cumm	300k/cumm	12	31.3	1.01mmom/l	0	
B/O Mamatha	3 months	Male	562134	Severe Sepsis in Shock	Not recordable	200/min	96	Gasping	Bilateral Equal and reactive	GCS 6/15	6.7	9	9.47	60	7.4	28	5.3	15	0.6	22.3k/cumm	387k/cumm	11.3	34.3	3.8mmol/l	21	Death
					not recordable	198/min	97	On Mv	Bilateral pupils dilted and fixed	GCS 5/15	6.78	14	10.2	54	10.2	90	6.9	15	0.6	24k/cumm	222k/cumm	12.7	33.7	4.2mmol/l	26	
Rohan	9 years	Male	568941	Diabetes Ketoacidosis	76/60	190/min	98	42/min	Bilateral Equal and reactive	GCS 13/15	6.98	11	17.64	120	11.2	960	4.5	4.5	0.4	21.62k/cumm	412k/cumm	16.3	46.4	2.8mmol/l	11	Recovered
					88/60	166/min	98	40/min	Bilateral Equal and reactive	GCS 14/15	7.03	15	16.25	122	12.8	234	4.5	4.2	0.6	18k/cumm	400k/cumm	13.4	39.3	2.6mmol/l	8	
					92/60	140/min	98	36/min	Bilateral Equal and reactive	GCS 14/15	7.21	22	20.3	130	15.3	200	4	4	0.6	6.8k/cumm	400k/cumm	17.3	35.2	1.7mmol/l	2	
Shaikh Afrid Uddn	12 years	Male	562147	Severe Dengue fever	70/40	120/min	98	120/min	Bilateral Equal and reactive	GCS 15/15	7.21	15	21.6	100	18.2	98	4.2	15	0.4	8.78K/cumm	169/cumm	14.5	42.3	1.1mmol/l	8	Recovered
					80/40	110/min	99	110/min	Bilateral Equal and reactive	GCS 15/15	7.24	34	27.8	102	20.3	112	4.2	15	0.4	5.89/cumm	90k/cumm	23.1	36.3	1mmol/l	5	
					96/54	98/min	102	98/min	Bilateral Equal and reactive	GCS 15/15	7.24	34	30.4	102	22.6	112	4.3	15	0.4	6k/cumm	58k/cumm	22.1	37.3	0.8mmol/l	5	
Anusha	13 years	Female	574123	Respiratory Failure	84/60	170/min	98	54/min	Bilateral Equal and reactive	GCS 10/15	7.18	21	21.7	60	16.2	98	4.8	15	0.4	28k/cumm	450/cumm	20.4	67.5	2.2mmol/l	12	Recovered
					90/60	120	99	52/min	Bilateral Equal and reactive	GCS 15/15	7.24	32	26.6	90	19.3	96	4.2	20	0.5	22k/cumm	325k/cumm	15.4	47.3	2.1mmol/l	5	
					92/60	110/min	98	42/min	Bilateral Equal and reactive	GCS 15/15	7.24	35	29.6	85	21.6	102	4.4	13	0.5	20k/cumm	42k/cum	16.3	42.4	2.01mmol/l	5	
Nafiza	6months	Female	521036	Acute GE with severe dehydration	not recordable	190/min	96	68/min	Bilateral Equal and reactive	GCS 10/15	6.9	14	13.52	54	10.3	20	2.6	21	1	19.9k/cumm	1147k/cumm	12.4	47.4	3.4mmol/l	13	Recovered
					not recordable	201/min	96	72/min	Bilateral pupils dilted and fixed	GCS 12/15	6.8	24	10.9	52	8.6	60	4.2	22	1.3	28k/cumm	420k/cumm	13.6	43.4	3mmol/l	13	
					60/42	201/min	96	72/min	Bilateral Equal and reactive	GCS 10/15	6.9	28	13.04	50	11.2	92	4.2	30	1.5	28k/cumm	378k/cumm	15.8	38.3	3.3mmol/l	5	
Akul	9 years	Male	528974	Diabetes Ketoacidosis	100/60	98/min	98	24/min	Bilateral Equal and reactive	GCS 15/15	7.26	18	18.5	68	18.3	HIGH	5.6	13	0.6	20.86k/cumm	566k/cumm	16.2	47.2	1.3mmol/l	4	Recovered
					100/60	9/min	96	24/min	Bilateral Equal and reactive	GCS 15/15	7.35	28	26.8	75	20.4	526	5.4	24	0.8	10k/cumm	428k/cumm	18.2	46.3	1mmol/l	4	
					98/69	92/min	96	26/min	Bilateral Equal and reactive	GCS 15/15	7.4	35	30.4	106	22.4	192	5.4	13	0.4	14k/cumm	420k/cumm	13.5	33.6	1mmol/l	2	
Varshini	12 months	Female	521031	Acute GE with severe dehydration	52/40	190/mn	96	48/min	Bilateral Equal and reactive	GCS 10/15	7.18	23	27.6	78	12.3	24	4.6	24	1.2	22k/cumm	483k/cumm	14.3	37.3	2.2mmol/l	17	Recovered
					62/40	162/min	98	38/min	Bilateral Equal and reactive	GCS 12/15	7.24	30	21.5	96	14.6	92	4.2	20	0.9	20k/cumm	450k/cumm	13.2	33.5	1.8mmol/l	7	
					70/50	140/min	98	34/min	Bilateral Equal and reactive	GCS 15/15	7.35	32	27.6	106	20.3	98	4	18	0.6	12k/cumm	428k/cumm	12.3	36.7	1.5mmol/l	7	
Chaithanya	15 years	Female	586320	Dengue Fever with warning signs	72/50	130/min	97	40/min	Bilateral Equal and reactive	GCS 15/15	7.38	38	33.7	100	22.3	98	4.5	14	0.4	28k/cumm	208k/cumm	19.8	32.4	2.2mmol/l	3	Recovered
					90/50	120/min	99	34/min	Bilateral Equal and reactive	GCS 15/15	7.45	37	32.8	108	24.2	90	4.3	12	0.6	12k/cumm	200k/cumm	17.3	33.2	1.5mmol/l	0	
					92/60	98/min	99.2	28/min	Bilateral Equal and reactive	GCS 15/15	7.46	38	34.04	120	25.3	92	4.2	14	0.3	21K/CUMM	214k/cumm	17.8	33.4	1.6mmol/l	0	
Santhosh	15 years	Male	541206	Diabetes Ketoacidosis	76/40	122/min	99	48/min	Bilateral Equal and reactive	GCS 15/15	6.89	16.2	14.3	132	10.6	HIGH	7.8	17	0.4	65K/CUMM	698k/cumm	12.3	43.2	2.6mmol/l	17	Recovered
					98/50	120/min	99	36/min	Bilateral Equal and reactive	GCS 15/15	7.16	17.4	15.9	120	12.3	283	4.1	17	0.4	21K/CUMM	693k/cumm	13.2	37.4	1.8mmol/l	10	
					100/60	110/min	99	32/min	Bilateral Equal and reactive	GCS 15/15	7.23	22	23.36	130	18.3	330	4.3	6	0.4	13K/CUMM	550k/cumm	24.5	39.3	1.5mmol/l	10	
Rayan	4 months	Male	521036	Acute GE with severe dehydration	Not recordable	170/min	96	60/min	Bilateral Equal and reactive	GCS 10/15	6.9	19	16.94	56	8.2	18	3.4	46	0.8	28k/cumm	238k/cumm	21.1	38.4	3.1mmol/l	16	Recovered

Name	Age	Sex	IP number	Diagnosis	BP	HR	Temperature	RR	Pupils	GCS	pH	pCO2	Total CO2	PaO2	Bicarbonate	Glucose	Potassium	BUN	Creatinine	WBC	Platelet	PT	PTT	Serum Lactate	PRISM III	Out Come
					56/40	160/min	98	68/min	Bilateral Equal and reactive	GCS 12/15	7.12	26	16.67	62	12.3	82	3.4	46	1.2	32k/cumm	204k/cumm	19.5	36.3	2.7mmol/l	8	
					60/40	142/min	98	60/min	Bilateral Equal and reactive	GCS 13/15	7.3	28	24.4	64	18.3	106	3.2	40	1.3	38k/cumm	200k/cumm	14.3	42.2	2.3mmol/l	8	
Mohammad Farhan	2 months	Male	521036	Acute GE with severe dehydration	not recordable	190/min	96	72/min	Bilateral Equal and reactive	GCS 10/15	6.8	14.9	13	193	10.3	20	5.6	32	1.6	33k/cumm	530k/cumm	12.5	46.2	3.4mmol/l	18	Recovered
					52/40	160/min	98	On Mv	Bilateral Equal and reactive	GCS 9/15	6.9	15	12.2	120	9.2	92	6	33	1.8	36k/cumm	420k/cumm	13.2	43.3	3.7mmol/l	18	
					62/40	178/min	97	On Mv	Bilateral Equal and reactive	GCS 8/15	6.8	12	16.8	118	14.2	96	6.2	42	2	40k/cumm	400k/cumm	15.6	39.2	4mmol/l	11	
Isaan ahmed	2 months	Male	547896	Acute GE with severe dehydration	not recordable	190/min	96	64/min	Bilateral Equal and reactive	GCS 11/15	7.1	20	20.3	120	16.3	38	4.2	15	0.4	14k/cumm	549k/cumm	19.8	47.1	3.1mmol/l	12	Recovered
					58/40	176/min	97	48/min	Bilateral Equal and reactive	GCS 12/15	7.28	30	24.2	124	18.2	92	4.3	16	0.5	10k/cumm	455k/cumm	17.5	45.3	2.6mmol/l	10	
					62/40	154/min	98	40/min	Bilateral Equal and reactive	GCS 14/15	7.35	38	27.3	130	20.3	102	4	16	0.5	10k/cumm	400k/cumm	15.3	33.5	2.1mmol/l	8	
Chandana	13 years	Female	512036	Acute GE with severe dehydration	80/60	198/min	98	24/min	Bilateral Equal and reactive	GCS 10/15	6.9	16	13.5	98	10.5	96	4.2	40	1.8	48k/cumm	480k/cumm	17.2	33.7	3.1mmol/l	18	Recovered
					82/60	172/min	98	22/min	Bilateral Equal and reactive	GCS 12/15	7.12	20	16.3	96	12.3	120	4	42	1.6	42k/cumm	420k/cumm	16.5	36.3	2.7mmol/l	18	
					90/60	120/min	98	24/min	Bilateral Equal and reactive	GCS 15/15	7.34	35	25.2	98	18.2	128	3.3	44	1.2	36k/cumm	400k/cumm	23.3	32.2	2mmol/l	9	
Thanushree	5 years	Female	512036	Acute Dengue fevere withARDS	74/60	112/min	96	36/min	Bilateral Equal and reactive	GCS 10/15	7.12	30	18.3	100	12.3	128	2.9	25	0.5	14.71k/cumm	45k/cumm	18.5	44.6	2.35mmol/l	8	Recovered
					92/60	120/min	98	38/min	Bilateral Equal and reactive	GCS 99/15	7.1	22	17.4	53	13.4	124	3.8	26	0.6	29.09k/cumm	19k/cumm	19.6	42.3	2.5mmol/l	8	
					90/90	136/min	96	38/min	Bilateral Equal and reactive	GCS 12/15	7	12	12.4	43	10.2	100	3.9	12	0.6	8.61k/cumm	126k/cumm	10.3	38.6	2.1mmol/l	5	
Aman	10years	Male	52961	Type I Diabetes Mellitus	86/70	110/min	98	30/min	Bilateral Equal and reactive	GCS 15/15	7.4	10.2	26.3	72	24.3	HIGH	6.3	27	0.5	17250/cumm	328000/cumm	11.6	36.6	2mmol/l	4	Recovered
				Diabetes Ketoacidosis	92/60	122/min	98	32/min	Bilateral Equal and reactive	GCS 15/15	7.2	16.6	25.8	149	22.6	518	3.1	18	0.7	12000/cumm	328k/cumm	13.2	33.4	1.8mmol/l	2	
					94/60	94/min	99	28/min	Bilateral Equal and reactive	GCS15/15	7.36	22.6	30.2	94	26.2	198	3	14	0.4	12000/cumm	328k/cumm	15.3	32.3	2mmol/l	2	
Shyni	5 years	Female	54326	Diabetes Ketoacidosis	78/50	118/min	99	50/min	Bilateral Equal and reactive	GCS 15/15	7.08	6	13.6	127.6	12.4	HIGH	4.7	21	0.5	30.09k/cumm	445k/cum	14.3	48.3	2mmol/l	7	Recovered
					80/54	120/min	99	42/min	Bilateral Equal and reactive	GCS 15/15	7.24	11.8	18.7	155	16.3	430	3.4	13	0.3	5.36k/cumm	318k/cumm	17.5	38.5	1.6mmol/l	4	
					84/60	124/min	99	36/min	Bilateral Equal and reactive	GCS 15/15	7.36	20.9	22.6	108	18.2	182	2.9	12	0.2	8k/cumm	300k/cumm	18.4	36.2	1.4mmol/l	2	
Balaji	15 years	Male	543114	Diabetic ketoacidosis	100/60	136/min	99	46/min	Bilateral Equal and reactive	GCS 10/15	7	19	17	74	13.2	High	3.9	23	1.8	22k/cumm	605k/cumm	12.4	47.2	2.3mmol/l	9	Recovered
					102/60	160/min	98	48/min	Bilateral Equal and reactive	GCS 13/15	7.36	14.2	18.1	82	15.3	447	4.6	12	1.4	26k/cumm	420/cumm	13.2	33.3	2mmol/l	9	
					120/60	154/min	99	36/min	Bilateral Equal and reactive	GCS 15/15	7.44	22	22.4	100	18.2	250	4.9	10	0.8	20k/cumm	400k/cumm	11.3	32.4	1.5mmol/l	7	
Sai Shravani	17 years	Female	541731	Diabetic ketoacidosis	110/60	110/min	99	38/min	Bilateral Equal and reactive	GCS 15/15	6.9	13.9	12.8	76	10.2	494	3	15	0.7	21k/cumm	507k/cumm	13.6	38.6	3.2mmol/l	11	Recovered
					110/60	118/min	98	36/min	Bilateral Equal and reactive	GCS 15/15	7.24	15.8	20.1	80	16.2	308	2.6	13	0.6	16k/cumm	420k/cumm	12.4	43.3	2.6mmol/l	6	
					110/60	98/min	99	32/min	Bilateral Equal and reactive	GCS 15/15	7.33	16	23.4	84	20.3	190	4	12	0.8	10k/cumm	408k/cumm	16.5	36.6	2.2mmol/l	4	
Nandakumar	17yrs	Male	625688	Diabetes Ketoacidosis	96/60	106/min	99	42/min	Bilateral Equal and reactive	GCS 15/15	7.1	16.3	15.3	83	12.3	HIGH	5.6	12	0.6	21K/CUMM	250k/cumm	13.4	48.5	2.9mmol/l	10	Recovered
					100/60	108/min	98	35/min	Bilateral Equal and reactive	GCS 15/15	7.25	20.2	20.6	80	16.3	420	4.2	11	0.6	15k/cumm	215k/cumm	12.3	32.2	2.1mmol/l	8	
					100/60	100/min	99	30/min	Bilateral Equal and reactive	GCS 15/15	7.35	32.03	28.4	76	22.4	356	4.5	12	0.8	14k/cumm	210k/cumm	11.3	36.5	1.6mmol/l	6	
B/o Pooja	3 months	Male	625768	Acute GE with severe dehydration	not recordable	180/min	97	78/min	Bilateral Equal and reactive	GCS 12/15	6.9	18.2	11.2	86	8.8	156	6.8	56	2	25k/cumm	450k/cumm	14.3	33.6	2.8mmol/l	14	Recovered
					65/40	176/min	99	68/min	Bilateral Equal and reactive	GCS 13/15	7.05	28.3	17.3	88	12.3	152	6.2	52	1.6	20k/cumm	425k/cumm	12.4	32.4	2.2mmol/l	10	
					72/40	152/min	99.2	62/min	Bilateral Equal and reactive	GCS 15/15	7.12	32.1	20.2	100	13.2	145	5.8	45	1.8	22k/cumm	350k/cumm	11.6	35.4	1.6mmol/l	8	
Lekha	7 months	female	629203	Bronchopneumonia , Sepsis in Shock	52/30	176/min	102	78/min	Bilateral Equal and reactive	GCS12/15	6.9	16	11.8	86	8.2	250	7	32	2.1	12k/cumm	250k/cumm	13.4	45.3	3.1mmol/l	17	Recovered
					65/40	188/min	100	84/min	Bilateral Equal and reactive	GCS 13/15	7.15	12.02	15.8	92	13.4	150	6.5	15	1.68	16k/cumm	265k/cumm	16.1	38.5	2.5mmol/l	9	
					72/40	165/min	101	76/min	Bilateral Equal and reactive	GCS 13/15	7.2	25	23.2	100	18.2	106	6.2	13	1.2	10k/cumm	270k/cumm	12.3	34.4	2.2mmol/l	6	
Teja Rao	3 months	Male	622944	Bronchopneumonia , Sepsis in Shock	not recordable	186/min	96	On MV	Bilateral Equal and reactive	GCS 8/15	6.8	9	9	45	7.2	120	6.8	30	2.5	32k/cumm	262k/cumm	12.3	36.2	3.8mmol/l	18	Death
					62/40	190/min	98	On MV	Bilateral Equal and reactive	GCS 7/15	6.9	18	13.8	106	10.2	122	6.9	32	2.3	28k/cumm	150k/cumm	12.3	37.3	4.3mmol/l	25	
					65/40	186/min	99.2	On MV	Bilateral Equal and reactive	GCS 8/15	6.96	20	13.9	108	9.3	112	6.5	33	2.4	26k/cumm	98k/cumm	14.5	35.4	4.5mmol/l	26	
B/o Devi	3 months	Male	622989	Bronchopneumonia , Sepsis in Shock	not recordable	210/min	96	On Mv	Bilateral Equal and reactive	GCS 7/15	6.85	10	12.2	42	10.2	26	5.8	45	2.5	15k/cumm	75k/cumm	12.4	37.3	3.9mmol/l	24	Death
					not recordable	200/min	99	On Mv	Bilateral pupils dilted and fixed	GCS 8/15	6.92	10	14.6	65	11.6	98	5.4	46	2.1	13k/cumm	80k/cumm	14.3	33.5	4.02mmol/l	18	
Nikhil	4 yrs	Male	683155	Dengue Fever with warning signs	72/56	115/min	102	32/min	Bilateral Equal and reactive	GCS 15/15	7.35	32	24.6	102	18.4	98	4.2	12	0.8	11k/cumm	75k/cumm	12.5	36.6	1.2mmol/l	10	Recovered
					80/60	112/min	101	28/min	Bilateral Equal and reactive	GCS 15/15	7.35	32	24.6	102	18.4	92	4.3	12	0.8	12k/cumm	65k/cumm	16.3	37.2	1.28mmol/l	8	
					86/60	102/min	102	26/min	Bilateral Equal and reactive	GCS 15/15	7.35	32	24.6	102	18.4	96	4.3	12	0.8	6k/cumm	54k/cumm	17.4	32.2	1mmol/l	8	
B/o Krishna veni	1 month	female	629998	Sepsis with septic shock	not recordable	192/min	97	On Mv	Bilateral pupils dilted and fixed	GCS 8/15	6.88	9	10	52	9.2	52	6.3	48	2.1	23k/cumm	98k/cumm	13.2	36.7	3.86mmol/l	18	Death
					not recordable	196/min	98	On Mv	Bilateral pupils dilted and fixed	GCS 7/15	6.92	10	14.6	63	12.6	98	6.9	52	2.2	26k/cumm	95k/cumm	14.2	35.5	3.92mmol/l	26	
Hanumanth Reddy	2months	Male	623875	Bronchopneumonia , Sepsis in Shock	not recordable	203/min	97	On Mv	Bilateral pupils dilted and fixed	GCS 7/15	6.85	12	14.5	38	11.8	96	7.2	56	2.6	15k/cumm	56k/cumm	15.2	43.3	4.12mmol/l	32	Death
					not recordable	206/min	97.6	On Mv	Bilateral pupils dilted and fixed	GCS 7/15	6.93	16	15.9	63	12.2	109	6.9	54	2.5	21K/CUMM	65k/cumm	16.4	35.4	4.5mmol/l	26	

Name	Age	Sex	IP number	Diagnosis	BP	HR	Temperature	RR	Pupils	GCS	pH	pCO2	Total CO2	PaO2	Bicarbonate	Glucose	Potassium	BUN	Creatinine	WBC	Platelet	PT	PTT	Serum Lactate	PRISM III	Out Come
Deekshith	5 years	Male	623950	Severe Sepsis in Shock	68/45	152/min	98	42/min	Bilateral Equal and reactive	GCS 13/15	7.12	16	22.28	75	18.6	120	6.2	25	1.8	15k/cumm	98k/cumm	16.3	37.6	2.6mmol/l	7	Recovered
					72/50	148/min	99.6	38/min	Bilateral Equal and reactive	GCS 14/15	7.26	25	26.05	150	20.3	103	5.6	20	1.5	26k/cumm	105k/cumm	18.9	33.6	2.2mmol/l	7	
					86/50	135/min	101	36/min	Bilateral Equal and reactive	GCS 15/15	7.36	32	30.6	128	22.4	98	5.3	18	1.2	14k/cumm	125k/cumm	13.5	33.5	1.9mmol/l	5	
B/o Mathin	3 months	Male	626793	Bronchopneumonia , Sepsis in Shock	not recordable	210/min	98.6	76/min	Bilateral Equal and reactive	GCS 13/15	7.01	25	18.9	78	13.2	96	5.6	32	1.6	15k/cumm	150k/cumm	23.2	32.3	2.8mmol/l	14	Recovered
					65/40	201/min	99.5	78/min	Bilateral Equal and reactive	GCS 15/15	7.21	26	24.38	102	18.4	102	5.3	28	1.3	12k/cumm	154k/cumm	21.4	35.4	2.6mmol/l	8	
					68/43	188/min	99.2	72/min	Bilateral Equal and reactive	GCS 15/15	7.32	30	27.5	130	20.6	106	4.9	29	0.9	13k/cumm	120k/cumm	20.5	36.3	2.1mmol/l	8	
Sandhya	1yr 5m	Female	627532	Aspiration Pneumonia	not recordable	65/min	97	On Mv	Bilateral pupils dilted and fixed	GCS 7/15	6.86	12	12.3	54	10.3	98	4.6	30	1.5	12k/cumm	210k/cumm	13.2	38.5	4.01mmol/l	33	Death
					not recordable	106/min	98.5	On Mv	Bilateral pupils dilted and fixed	GCS 7/15	6.94	16	12.88	106	9.2	96	4.6	26	1.6	10k/cumm	234k/cumm	13.4	40.3	4.3mmol/l	34	
Abhimanya	4 months	Female	629093	Sepsis with septic shock	not recordable	210/min	97	On Mv	Bilateral pupils dilted and fixed	GCS 7/15	6.95	16	14.2	92	10.6	102	6.8	42	2.5	25k/cumm	345k/cumm	14.3	44.5	3.9mmol/l	34	Death
					not recordable	206/min	96	On Mv	Bilateral pupils dilted and fixed	GCS 7/15	6.94	12	12.26	94	9.5	150	6.9	43	2.1	25k/cumm	345k/cumm	15.6	38.4	4.mmol/l	37	
Shreya Vaishanavi	13 yrs	female	628108	Diabetes Ketoacidosis	96/60	102/min	99.3	45/min	Bilateral Equal and reactive	GCS 15/15	6.98	16	12.28	98	8.6	HIGH	4.5	16	1.3	16k/cumm	210k/cumm	14.3	40.8	2.56mmol/l	13	Recovered
					100/60	106/min	99.6	40/min	Bilateral Equal and reactive	GCS 15/15	7.1	23	15.79	106	10.5	450	4.6	13	1.2	12k/cumm	265k/cumm	12.4	42.3	2.3mmol/l	6	
					100/60	108/min	98.6	26/min	Bilateral Equal and reactive	GCS 15/15	7.3	31.3	21.7	120	14.6	182	4.3	12	0.9	12k/cumm	234k/cumm	13.2	36.4	2.1mmol/l	4	
B/O Pallavi	1 month	Male	632176	Severe Sepsis in Shock	not recordable	210/min	97	On Mv	Bilateral pupils dilted and fixed	GCS 7/15	6.78	8.6	11.3	42	9.4	160	6.5	54	2.5	26k/cumm	120k/cumm	12.1	45.3	4.5mmol/l	34	Death
					not recordable	234/min	98	On MV	Bilateral pupils dilted and fixed	GCS 7/15	6.8	14.3	15.58	52	12.3	124	6.1	60	1.9	25k/cumm	163k/cumm	15.6	48.3	4.3mmol/l	34	
					not recordable	196/min	98	On Mv	Bilateral pupils dilted and fixed	GCS 7/15	6.82	9.2	12.76	62	10.6	150	5.9	58	2.1	28k/cumm	98k/cumm	13.4	40.5	4.35mmol/l	36	
B/O Sirisha	3 months	Male	637249	Severe Sepsis in Shock	not recordable	180/min	99.3	On Mv	Bilateral Equal and reactive	GCS 8/15	7.01	12.6	17.09	56	14.2	165	4.6	32	2.3	23k/cumm	120k/cumm	11.3	33.5	3.98mmol/l	30	Death
					not recordable	192/min	98.3	On MV	Bilateral pupils dilted and fixed	GCS7/15	7	18.3	19.5	53	15.3	126	4.3	30	1.2	23k/cumm	145k/cumm	12.3	36.3	4.1mmol/l	30	
					not recordable	172/min	99.5	On Mv	Bilateral pupils dilted and fixed	GCS 9/15	7.06	18.2	20.1	99	16	126	5.2	26	1.6	16k/cumm	96k/cumm	17.4	46.6	4.5mmol/l	23	
Devika	9 yrs	Female	641796	Diabetes Ketoacidosis	100/60	120/min	99.5	42/min	Bilateral Equal and reactive	GCS 15/15	7.06	15.6	20.8	105	17.3	HIGH	4.6	12	0.6	16k/cumm	235k/cumm	18.6	35.4	2.8mmol/l	4	Recovered
					102/60	126/min	98.6	36/min	Bilateral Equal and reactive	GCS 15/15	7.12	18.2	22.7	123	18.6	350	4.3	13	0.8	12k/cumm	210k/cumm	19	33.6	2.35mmol/l	2	
					100/60	120/min	99.2	29/min	Bilateral Equal and reactive	GCS 15/15	7.3	28.3	26.2	120	20.2	186	4.2	13	0.9	13k/cumm	265k/cumm	17.1	36.6	2.12mmol/l	2	
Bhoomika	16 years	Female	641821	Rat Poisoning with shock	80/40	132/min	102.3	38/min	Bilateral Equal and reactive	GCS 13/15	7.21	22.3	22.8	65	18.2	46	6.5	56	2.1	12k/cumm	98k/cumm	16.4	43.5	3.8mmol/l	10	Death
0					not recordable	140/min	99.2	36/min	Bilateral Equal and reactive	GCS 14/15	7.12	12.3	19.1	56	16.5	62	6.3	45	1.9	16k/cumm	120k/cumm	15.6	68.3	4.2mmol/l	10	
					76/50	126/min	101.5	42/min	Bilateral Equal and reactive	GCS 15/15	7.06	21.6	17.2	98	12.6	98	6.9	54	2.23	13k/cumm	95k/cumm	14.5	44.3	2.52mmol/l	13	
Ananya	3yrs	Female	641827	Bronchopneumonia , Sepsis in Shock	not recordable	180/min	99.6	On Mv	Bilateral Equal and reactive	GCS 10/15	7.1	19.3	15.1	32	10.6	145	4.6	21	1.2	16k/cumm	275k/cumm	13.6	33.4	3.5mmol/l	20	Recovered
					72/50	168/min	102.6	On Mv	Bilateral Equal and reactive	Sedated	7.06	21.6	17.5	102	13.2	126	4.9	16.3	0.9	21K/CUMM	345k/cumm	13.5	35.4	3.12mmol/l	14	
					84/56	156/min	100.3	On Mv	Bilateral Equal and reactive	Sedated	7.2	26.8	20.8	115	15.2	102	5.1	13	0.9	14k/cumm	235k/cumm	12.6	33.2	2.8mmol/l	14	
B/O Sirisha	6 month	female	638740	Bronchopneumonia , Sepsis in Shock	not recordable	186/min	99.6	On Mv	Bilateral Equal and reactive	GCS 12/15	6.98	18.3	13.8	27	10.3	165	6.2	26	1.6	17k/cumm	345k/cumm	11.7	47.3	4.1mmol/l	24	Death
					68/44	178/min	98.3	On Mv	Bilateral Equal and reactive	Sedated	7.08	24.6	20.8	76	15.7	123	6.3	28	1.8	23k/cumm	265k/cumm	18.6	57.7	4.2mmol/l	13	
					74/46	168/min	101.6	On Mv	Bilateral Equal and reactive	Sedated	7.2	28.3	20.3	92	14.3	108	5.9	24	1.5	19k/cumm	234k/cumm	13.6	67.7	4.03mmol/l	13	
B/O Varalakshmi	3 month	female	638743	Bronchopneumonia , Sepsis in Shock	not recordable	192/min	102.3	On Mv	Bilateral Equal and reactive	GCS 9/15	6.96	16.2	12.1	39	8.3	98	5.6	56	1.8	21K/CUMM	98k/cumm	14.3	34.5	5.02mmol/l	24	Recovered
					66/42	186/min	101.3	On Mv	Bilateral Equal and reactive	Sedated	6.68	21.3	8.9	78	4.6	102	5.8	48	1.5	16k/cumm	95k/cumm	15.7	32.3	4.35mmol/l	17	
					68/44	170/min	100.8	On Mv	Bilateral Equal and reactive	Sedated	7.06	22.3	21.3	82	16	106	4.9	36	0.9	18k/cumm	120k/cumm	13.2	33.5	4.2mmol/l	13	
Tejas	3year	Male	689301	Drowning	Not recordable	68/min	97	On Mv	Bilateral Equal and reactive	GCS 7/15	6.98	16.9	13.7	36	9.3	63	5.2	32	1.2	15k/cumm	210k/cumm	18.5	44.6	3.6mmol/l	23	Death
					Not recordable	110/min	96.8	On Mv	Bilateral pupils dilted and fixed	GCS 7/15	6.86	17.8	13.8	58	10.2	102	4.9	24	0.8	14k/cumm	234k/cumm	12.3	67.7	4.1mmol/l	32	
Akshay	3 years	Male	643113	Severe Sepsis in Shock	Not recordable	188/min	97.3	On Mv	Bilateral Equal and reactive	GCS 8/15	6.98	12.3	10.8	38	8.2	106	5.9	56	1.8	23k/cumm	456k/cumm	11.2	68.7	3.9mmol/l	24	Death
					Not recordable	186/min	96.3	On MV	Bilateral Equal and reactive	GCS 9/15	6.92	16.3	13.2	56	9.6	108	5.7	54	1.2	26k/cumm	450k/cumm	12.6	56.3	4mmol/l	22	
					72/42	178/min	98.3	On Mv	Bilateral Equal and reactive	GCS 8/15	6.86	9.6	10.8	65	8.5	109	6.3	58	2.1	16k/cumm	436k/cumm	12.1	64.6	4.2mmol/l	20	
B/O Naziya	1yr 5 m	Female	649093	Acute GE with severe dehydration	Not recordable	176/min	98.3	68/min	Bilateral Equal and reactive	GCS 12/15	6.98	18.3	13.8	96	9.2	32	5.3	26	1.8	12k/cumm	345k/cumm	14.3	56.6	3.1mmol/l	18	Recovered
					66/46	168/min	99.6	56/min	Bilateral Equal and reactive	GCS 14/15	7.12	25.3	20.8	120	15.2	116	5.6	28	1.5	13K/CUMM	215k/cumm	15.1	54.3	2.86mmol/l	14	
					72/52	156/min	100.3	54/min	Bilateral Equal and reactive	GCS 15/15	7.35	32.2	26.4	137	20.3	120	4.9	32	1.3	7k/cumm	265k/cumm	14.1	44.5	2.2mmol/l	11	
Ganavi	4 months	Female	645524	Severe Sepsis in Shock	Not recordable	198/min	97.3	On MV	Bilateral Equal and reactive	GCS 10/15	6.89	10.3	11.8	65	9.6	35	5.9	45	2.1	4k/cumm	96k/cumm	15.2	67.5	3.86mmol/l	22	Recovered
					64/42	184/min	98.5	On Mv	Bilateral Equal and reactive	Sedated	7.01	18.3	20.7	88	16.2	78	6.3	46	1.8	6k/cumm	102k/cumm	11.2	68	3.5mmol/l	12	
					68/44	176/min	99.6	On Mv	Bilateral Equal and reactive	Sedated	7.25	25.6	19.3	102	14.2	98	5.8	38	1.6	10k/cumm	120k/cumm	14.2	45	3.2mmol/l	12	
Arbiya	3 months	Female	645551	Bronchopneumonia , Sepsis in Shock	Not recordable	190/min	97.3	On Mv	Bilateral Equal and reactive	GCS 7/15	6.86	9.6	14.2	32	12.1	25	5.2	45	2.5	18k/cumm	54k/cumm	15.6	68.8	4.3mmol/l	33	Death
					Not recordable	186/min	97.6	On Mv	Bilateral pupils dilted and fixed	GCS 8/15	6.83	12.3	11.2	58	9.2	102	4.8	52	2.6	23k/cumm	68k/cumm	12.4	70.6	5.01mmol/l	33	

Name	Age	Sex	IP number	Diagnosis	BP	HR	Temperature	RR	Pupils	GCS	pH	pCO2	Total CO2	PaO2	Bicarbonate	Glucose	Potassium	BUN	Creatinine	WBC	Platelet	PT	PTT	Serum Lactate	PRISM III	Out Come
Janani	4months	Female	645524	Severe Sepsis in Shock	Not recordable	195/min	97.6	On Mv	Bilateral Equal and reactive	GCS 9/15	6.98	11.3	10.6	75	8.2	65	5.3	62	1.8	34k/cumm	87k/cumm	12.3	65.5	4.3mmol/l	18	Death
					66/42	189/min	98.3	On Mv	Bilateral Equal and reactive	GCS 11/15	7.02	16.3	15.3	82	12.3	98	5.9	56	2.1	23.9k/cumm	108k/cumm	11.3	64.5	3.8mmol/l	7	
					68/44	176/min	99.2	On Mv	Bilateral Equal and reactive	Sedated	7.16	22.9	16.1	72	15.5	136	6.2	45	1.9	16.6k/cumm	120k/cumm	11.2	70	4.1mmol/l	7	
Shashank K R	17 years	Male	648305	Acute Encephalitis Syndrome	85/42	126/min	101.2	26/min	Bilateral Equal and reactive	GCS 15/15	7.25	26.5	25.3	116	19.2	120	4.5	26	0.8	3.06k/cumm	45k/cumm	12.7	56	2.8mmol/l	13	Recovered
				In Shock	92/50	134/min	98.5	28/min	Bilateral Equal and reactive	GCS 15/15	7.32	32.3	29.1	118	22.3	103	4.3	20	0.6	6.8k/cumm	32k/cumm	13.8	45	2.5mmol/l	7	
					96/54	106/min	99.6	24/min	Bilateral Equal and reactive	GCS 15/15	7.35	45.3	33.2	126	24.2	98	4.2	18	0.5	10.2k/cumm	56k/cumm	17.3	38	2.2mmol/l	2	
Lakshmi Devi	13 yrs	Female	648487	Severe Dengue Fever	Not recordable	136/min	102.3	56/min	Bilateral Equal and reactive	GCS 13/15	7.23	26.3	23.4	78	18.2	86	4.5	16	0.8	4.09k/cumm	67k/cumm	14.2	45.4	3.8mmol/l	13	Death
				ARDS	84/50	145/min	100.2	On Mv	Bilateral Equal and reactive	GCS 10/15	7.02	13.2	12.6	35	10.3	98	5.1	18	0.6	5.2k/cumm	34k/cumm	13.2	36.4	4mmol/l	10	
Jahnavi	8 years	Female	646790	Severe Dengue Fever	Not recordable	162/min	103.2	64/min	Bilateral Equal and reactive	GCS 12/15	7.16	12.3	15.3	86	12.4	96	3.6	12	0.6	3.65k/cumm	23k/cumm	13	33.4	3.4mmol/l	14	Death
					Not recordable	175/min	99.3	68/min	Bilateral Equal and reactive	GCS 10/15	7	25.3	19.4	56	14.3	95	3.9	13	0.5	4k/cumm	15k/cumm	13.6	36.3	3.8mmol/l	18	
					72/48	145/min	101.2	On Mv	Bilateral Equal and reactive	GCS 7/15	7.08	9.6	12.8	42	10.4	102	4.2	16	0.4	3.45k/cumm	12k/cumm	19.2	67.5	4.01mmol/l	22	
Harshitha	4 years	Female	652178	Bronchopneumonia , Sepsis in Shock	74/42	152/min	102.3	78/min	Bilateral Equal and reactive	GCS 10/15	7.02	12.6	16.4	65	14.3	98	4.3	12	0.6	12.5k/cumm	120k/cumm	12.3	58	3.6mmol/l	7	Recovered
					76/40	165/min	101.9	75/min	Bilateral Equal and reactive	GCS 13/15	7.18	23.6	20.4	98	15.3	65	4.2	16	0.8	13.8k/cumm	108k/cumm	11.2	36.8	3.06mmol/l	7	
					80/50	146/min	100.3	64/min	Bilateral Equal and reactive	GCS 13/15	7.26	36.5	26.7	102	19.4	105	4.9	18	0.7	23.9k/cumm	210k/cumm	12.1	33.2	2.8mmol/l	5	
Nanda Kumar	11 years	Male	652683	Severe Dengue Fever	not recordable	152/min	99.3	64/min	Bilateral Equal and reactive	GCS 13/15	7.1	26.3	15.7	86	10.8	109	4.3	12	0.8	3.6k/cumm	23k/cumm	21.3	70.5	3.2mmol/l	10	Death
				ARDS	Not recordable	145/min	98.2	On Mv	Bilateral Equal and reactive	GCS 12/15	6.9	13.6	11.8	35	9.2	152	4.2	10	0.3	4.2k/cumm	9k/cumm	12.8	67.4	3.9mmol/l	24	
Sneha	13 years	Female	648802	Dengue Fever with warning signs	84/60	132/min	100.3	28/min	Bilateral Equal and reactive	GCS 15/15	7.12	20.3	17.8	102	13.2	102	4.2	12	0.8	4.3k/cumm	45k/cumm	12.1	37.5	2.6mmol/l	10	Recovered
					92/70	120/min	99.3	26/min	Bilateral Equal and reactive	GCS 15/15	7.23	28.6	20.8	108	15.3	98	4.3	13	0.7	5.6k/cumm	56k/cumm	12.6	38.7	2.1mmol/l	6	
					100/60	106/min	98.6	24/min	Bilateral Equal and reactive	GCS 15/15	7.42	46.3	31.2	125	22.3	96	4.8	11	0.5	6.3k/cumm	65k/cumm	11.2	33.2	1.8mmol/l	5	
Arshiya	5 years	Female	652871	Dengue Fever with warning signs	68/42	135/min	99.6	23/min	Bilateral Equal and reactive	GCS 15/15	7.26	35.6	19.2	85	12.6	96	4.3	11	0.6	3.2k/cumm	86k/cumm	12.6	56.6	2.1mmol/l	9	Recovered
					78/56	120/min	101.3	28/min	Bilateral Equal and reactive	GCS 15/15	7.36	38.3	30.2	92	23.2	102	4.2	12	0.8	3.6k/cumm	75k/cumm	12.7	46.4	1.8mmol/l	4	
					90/60	85/min	100	26/min	Bilateral Equal and reactive	GCS 15/15	7.38	40.2	29.3	89	21.5	95	3.8	18	0.4	12k/cumm	65k/cumm	13.1	35.5	1.6mmol/l	4	
Ayan Pasha	7 years	Male	657337	Dengue Fever with warning signs	74/45	102/min	102.3	25/min	Bilateral Equal and reactive	GCS 15/15	7.26	32.3	28.3	112	22.7	98	3.6	12	0.6	4.5k/cumm	67k/cumm	14.2	45.5	2.5mmol/l	9	Recovered
					88/60	96/min	99.3	24/min	Bilateral Equal and reactive	GCS 15/15	7.38	36.3	31.8	154	24.3	96	4.2	13	0.8	5.6k/cumm	54k/cumm	12.1	37.7	2mmol/l	4	
					90/60	88/min	98.6	22/min	Bilateral Equal and reactive	GCS 15/15	7.4	40.7	33.5	123	25.3	103	5.3	18	0.7	7.7k/cumm	87k/cumm	13.3	32.2	1.2mmol/l	4	
Yash	2 years	Male	653446	Acute GE with severe dehydration	not recordable	156/min	98.6	46/min	Bilateral Equal and reactive	GCS 14/15	7.01	12.3	12.8	84	10.5	102	4.5	12	0.6	6.7k/cumm	85k/cumm	14.2	43.4	3.6mmol/l	13	Recovered
					68/40	162/min	99.3	38/min	Bilateral Equal and reactive	GCS 15/15	7.12	20.3	17.9	92	13.8	136	4.6	13	0.8	5.6k/cumm	95k/cumm	12.3	36.4	2.8mmol/l	9	
					72/56	142/min	100.3	32/min	Bilateral Equal and reactive	GCS 15/15	7.22	28.6	19.5	100	14.6	126	4.3	15	0.7	10k/cumm	120k/cumm	12.8	33.5	3mmol/l	7	
Harishree	6 years	Female	661280	Dengue Fever with warning signs	74/60	125/min	102.3	26/min	Bilateral Equal and reactive	GCS 15/15	7.25	34.2	25.6	126	18.6	106	4.3	12	0.9	3.4k/cumm	67k/cumm	16.1	45.6	2.6mmol/l	9	Recovered
					82/64	98/min	98.6	24/min	Bilateral Equal and reactive	GCS 15/15	7.36	40	31.2	130	22.3	132	4.2	14	0.5	4.5k/cumm	88k/cumm	15.2	42.2	2.1mmol/l	6	
					90/60	84/min	99.3	22/min	Bilateral Equal and reactive	GCS 15/15	7.4	39	28.4	165	20.3	156	3.9	18	0.7	10k/cumm	90k/cumm	13.4	35.5	1.5mmol/l	6	
Gagan	5 years	Male	659761	Dengue Fever with warning signs	72/56	98/min	101.3	24/mn	Bilateral Equal and reactive	GCS 15/15	7.28	23.6	21.6	88	17.2	106	3.6	12	0.7	4k/cumm	56k/cumm	12.1	69.6	2.2mmol/l	9	Recovered
					82/60	96/min	100	26/min	Bilateral Equal and reactive	GCS 15/15	7.36	32.3	24.6	90	18.3	102	4.6	13	0.6	5k/cumm	67k/cumm	12.1	49.5	1.8mmol/l	4	
					90/60	88/min	99.3	23/min	Bilateral Equal and reactive	GCS 15/15	7.45	36.2	32.4	92	25.2	98	4.2	14	0.8	11k/cumm	89k/cumm	12.1	40.6	1.6mmol/l	4	
Purandareshwari	6 yrs	Female	660193	Severe Dengue Fever	not recordable	162/min	102.3	78/min	Bilateral Equal and reactive	GCS 12/15	7.15	16.3	16.5	84	13.4	96	5.9	56	2.2	21K/CUMM	15k/cumm	21.3	67.5	3.8mmol/l	19	Death
					78/50	168/min	100	On MV	Bilateral Equal and reactive	GCS 9/15	6.98	10.3	9.8	72	7	98	6.8	48	2.5	26k/cumm	12k/cumm	18.3	72.2	4.5mmol/l	19	
Srikanth	8 years	Male	662876	Dengue Fever with warning signs	78/56	126/min	100	30/min	Bilateral Equal and reactive	GCS 15/15	7.35	36.2	25.3	145	18.2	98	4.3	12	1.1	4.5k/cumm	45k/cumm	13.5	44.3	1.9mmol/l	7	Recovered
					92/60	130/min	102.3	28/min	Bilateral Equal and reactive	GCS 15/15	7.38	38.3	29.7	126	22.4	102	4.2	13	0.8	4k/cumm	89k/cumm	12.3	33.5	1.6mmol/l	7	
					94/60	122/min	98.6	26/min	Bilateral Equal and reactive	GCS 15/15	7.45	40	30.8	152	22.3	100	4.9	18	0.7	6k/cumm	120k/cumm	11.2	32.3	1.2mmol/l	4	
B/O Soundarya	1 month	Male	664766	Severe Sepsis in Shock	not recordable	198/min	96.3	On MV	Bilateral pupils dilted and fixed	GCS 8/15	6.98	22	13.8	56	9.6	26	6.2	56	2.1	2.6k/cumm	15k/cumm	18.6	67.5	4.02mmol/l	38	Death
					not recordable	186/min	97.3	On MV	Bilateral pupils dilted and fixed	GCS 7/15	6.97	31.3	16.2	86	10.6	92	6.5	54	1.9	5k/cumm	13k/cumm	19.3	72.2	4.3mmol/l	41	
Vikas Chandra	3 months	Male	665639	Severe Sepsis in Shock	not recordable	190/min	97	On MV	Bilateral Equal and reactive	GCS 9/15	6.86	20.3	12.7	98	8.7	96	4.6	20	1.2	23k/cumm	76k/cumm	17.4	56.4	3.9mmol/l	17	Death
					not recordable	185/min	99.3	On MV	Bilateral Equal and reactive	GCS 10/15	6.86	28.3	14.3	58	9.2	98	4.5	28	1.6	26k/cumm	152k/cumm	18.3	57.3	4.1mmol/l	17	
					not recordable	210/min	98.3	On MV	Bilateral pupils dilted and fixed	GCS 10/15	6.88	15.6	13.2	101	10.2	94	5.3	13	0.9	19k/cumm	92k/cumm	12.3	45.4	4.2mmol/l	18	
B/O Shyamala	3 months	Male	666871	Severe Sepsis in Shock	not recordable	203/min	97.3	On MV	Bilateral Equal and reactive	GCS 10/15	6.96	20.2	13.4	76	9.3	63	4.3	18	1.2	25k/cumm	98k/cumm	12.1	56.6	4.3mmol/l	23	Recovered
					66/46	195/min	99.6	On MV	Bilateral Equal and reactive	Sedated	7.12	23	16.8	96	12.3	88	4.2	25	1.4	20k/cumm	150k/cumm	13.2	46.6	3.6mmol/l	16	
					68/42	176/min	98.3	On MV	Bilateral Equal and reactive	Sedated	7.25	30.2	22.3	100	16.2	99	4.8	24	1.6	16k/cumm	156k/cumm	12.6	35.6	2.9mmol/l	16	

Name	Age	Sex	IP number	Diagnosis	BP	HR	Temperature	RR	Pupils	GCS	pH	pCO2	Total CO2	PaO2	Bicarbonate	Glucose	Potassium	BUN	Creatinine	WBC	Platelet	PT	PTT	Serum Lactate	PRISM III	Out Come
Suma	14 years	Female	668800	Diabetic ketoacidosis	96/60	120/min	98.6	54/min	Bilateral Equal and reactive	GCS 15/15	7.01	16.3	21.3	100	17.5	HIGH	4.6	25	0.8	21K/CUMM	234k/cumm	11.4	60.4	3.1mmol/l	7	Recovered
					98/60	112/min	99.3	42/min	Bilateral Equal and reactive	GCS 15/15	7.21	25.3	24.3	106	18.2	365	4.5	12	0.6	16k/cumm	256k/cumm	15.4	53.3	2.8mmol/l	4	
					100/60	102/min	99.2	32/min	Bilateral Equal and reactive	GCS 15/15	7.3	28.2	25.6	109	20.3	192	4.8	13	0.7	14k/cumm	150k/cumm	12.4	43.3	2.4mmol/l	4	
Nishanth	3 months	Male	671956	Severe Sepsis in Shock	not recordable	178/min	97.2	On Mv	Bilateral pupils dilted and fixed	GCS 8/15	6.86	12.3	12.8	68	10.8	26	5.3	58	2	3.2k/cumm	88k/cumm	18.6	67.4	4.05mmol/l	33	Death
					not recordable	180/min	98.3	On Mv	Bilateral pupils dilted and fixed	GCS 9/15	6.82	10.2	11.6	102	9.5	82	4.9	60	1.9	2.2k/cumm	76k/cumm	19.8	56.3	4.5mmol/l	33	
B/O mamatha	2 months	Male	675826	Severe Sepsis in Shock	not recordable	198/min	96.8	On Mv	Bilateral pupils dilted and fixed	GCS 11/15	6.92	18.3	14.2	65	10.6	39	4.2	58	1.8	25k/cumm	56k/cumm	24.2	72.3	4.2mmol/l	39	Death
					not recordable	186/min	98.6	On MV	Bilateral Equal and reactive	GCS 10/15	6.98	20.6	14.2	66	9.4	45	4.3	56	1.2	25k/cumm	28k/cumm	23.2	78.3	4.5mmol/l	39	
					not recordable	186/min	98.3	On Mv	Bilateral pupils dilted and fixed	GCS 10/15	6.84	12.3	13.2	102	10.4	106	4.6	45	1.62	27k/cumm	65k/cumm	16.3	65.4	4.6mmol/l	39	
Prashanth Reddy	17 years	Male	677305	Dengue Fever with warning signs	76/48	120/min	100.4	34/min	Bilateral Equal and reactive	GCS 15/15	7.28	34	22.4	103	16.2	120	3.8	12	1.2	4k/cumm	20k/cumm	12.3	33.4	2.1mmol/l	12	Recovered
					92/60	100/min	99	30/min	Bilateral Equal and reactive	GCS15/15	7.35	36	27.3	96	20.3	98	4.6	16	0.9	6.4k/cumm	9k/cumm	12.5	32.3	1.8mmol/l	8	
					100/60	80/min	99.3	24/min	Bilateral Equal and reactive	GCS15/15	7.4	38.2	31.5	140	24.2	97	4.9	11	0.7	10k/cumm	24k/cumm	12	35.3	1.2mmol/l	8	
B/O Lalitha	3m	Male	678145	Severe Sepsis in Shock	Not recordable	176/min	97.6	On MV	Bilateral Equal and reactive	GCS 8/15	6.9	11.2	23.2	40	10.3	41	6.8	56	2.1	21K/CUMM	234k/cumm	21.3	68.5	4.1mmol/l	18	Death
					not recordable	184/min	99.2	On Mv	Bilateral pupils dilted and fixed	GCS 7/15	7.12	20.3	18.5	86	14.2	127	5.2	60	1.9	18k/cumm	245k/cumm	22.4	72.2	4.5mmol/l	30	
Ganavi	7yr	Female	678116	Acute GE with severe dehydration	54/40	140/min	97.3	32/min	Bilateral Equal and reactive	GCS 14/15	7.12	24.2	22.3	76	16.2	79	6	46	1.1	24k/cumm	223k/cumm	13.4	43.3.	3.2mmol/l	10	Recovered
					82/58	132/min	98.7	28/min	Bilateral Equal and reactive	GCS 15/15	7.34	29.3	30.5	103	24.6	83	5.2	34	0.9	20.8k/cumm	256k/cumm	12.3	36.4	2.8mmol/l	3	
					88/60	120/min	96	26/min	Bilateral Equal and reactive	GCS 15/15	7.4	42.3	28.3	106	20.3	106	5	23	0.4	14k/cumm	343k/cumm	12.6	33.4	1.6mmol/l	3	
Charani	5yr	Female	678259	Acute GE with severe dehydration	not recordable	135/min	98.3	38/min	Bilateral Equal and reactive	GCS 13/15	7.02	23.2	16.8	90	12.3	98	4.7	40	1.2	15k/cumm	235k/cumm	12.6	44.5	2.6mmol/l	14	RECOVERED
					74/54	127/min	99.6	36/min	Bilateral Equal and reactive	GCS 14/15	7.18	27.5	19.7	98	14.3	128	4.2	32	1	12k/cumm	190k/cumm	12.4	34.5	1.8mmol/l	9	
					88/60	110/min	99.4	28/min	Bilateral Equal and reactive	GCS15/15	7.3	32.4	25.7	102	19.3	103	3.8	28	0.8	8k/cumm	138k/cumm	12.3	33.5	1.4mmol/l	0	
B/o Ashwini	9m	Male	678444	Acute GE with severe dehydration	not recordable	167/min	98.7	65/min	Bilateral Equal and reactive	GCS 12/15	6.9	18.3	12.3	82	7.6	140	6.2	36	1.1	32k/cumm	324k/cumm	11.2	43.6	2.8mmol/l	18	Recovered
					66/42	154/min	99.3	60/min	Bilateral Equal and reactive	GCS14/15	7.1	28.4	17.5	92	12.8	123	5.8	32	0.8	38k/cumm	423k/cumm	12	44.6	2.2mmol/l	5	
					72/50	132/min	100.1	56/min	Bilateral Equal and reactive	GCS 15/15	7.32	32.5	22.4	100	16.2	96	4.8	12	0.6	25k/cumm	367k/cumm	12.5	43.5	1.8mmol/l	0	
Kiran	2years	Male	678600	Acute GE with severe dehydration	not recordable	142/min	98.6	66/min	Bilateral Equal and reactive	GCS 13/15	6.8	16.3	15.8	126	12.5	23	6.3	56	1.8	23k/cumm	456k/cumm	11.3	46.5	3.1mmol/l	18	Recovered
					56/40	156/min	99.3	58/min	Bilateral Equal and reactive	GCS 15/15	7.1	28.5	21.8	102	16.4	98	5.6	48	1.5	19.6k/cumm	345k/cumm	12.6	47.6	2.73mmol/l	10	
					80/60	124/min	99.5	54/min	Bilateral Equal and reactive	GCS 15/15	7.28	32.9	22.3	98	16.5	103	4.4	34	1.3	16k/cumm	229k/cumm	12.7	36.4	1.8mmol/l	5	
Subhashini	13years	Female	679842	Severe Dengue	Not recordable	156/mn	96.4	63/min	Bilateral Equal and reactive	GCS 12/15	6.82	14.6	13.5	34	10.3	108	5.8	56	1.7	1.9k/cumm	4k/cumm	14.2	65.4	3.9mmol/l	33	Death
					not recordable	143/min	98.3	On MV	Bilateral pupils dilted and fixed	GCS 10/15	6.9	13.4	16.2	45	13.2	123	5.2	48	1.7	2.3k/cumm	9k/cumm	15.2	66.6	4.2mmol/l	44	
Simun	1yr 5 m	Male	680665	Bronchopneumonia , Sepsis in Shock	Not recordable	165/min	98.3	On MV	Bilateral Equal and reactive	GCS 8/15	6.8	21.2	12.3	47	7.6	17	6.3	63	1.2	24k/cumm	15k/cumm	12.3	49.5	3.2mmol/l	23	Recovered
					68/45	168/min	97.6	On MV	Bilateral Equal and reactive	GCS10/15	7	25.6	21.6	56	16.3	87	5.9	56	1.01	21k/cumm	59k/cumm	13.5	40.2	2.6mmol/l	14	
					72/50	154/min	99.6	On Mv	Bilateral Equal and reactive	GCS 13/15	7.23	32.1	24.8	78	18.4	80	5.5	48	0.8	18k/cumm	120k/cumm	14.2	34.4	2.3mmol/l	7	
Bhargav	1year	Male	680699	Bronchopneumonia , Sepsis in Shock	not recordable	170/min	98.3	On MV	Bilateral Equal and reactive	GCS 12/15	6.9	29	16.2	43	10.7	108	6.1	53	1.2	17k/cumm	342k/cumm	14.3	47.4	3.3mmol/l	21	Recovered
					68/43	166/min	99.6	On Mv	Bilateral Equal and reactive	GCS 13/15	7.12	34	18.7	83	12.4	120	5.3	42	0.9	21k/cumm	345k/cumm	17.4	44.3	2.5mmol/l	8	
					76/56	162/min	100.2	On Mv	Bilateral Equal and reactive	GCS 13/15	7.3	36	24.8	90	17.6	90	5.1	38	0.8	12k/cumm	347k/cumm	13.5	35.5	2.0mmol/l	3	
Zoya	7 years	Femal	680719	Severe Dengue Fever	Not recordable	134/min	97.3	On MV	Bilateral Equal and reactive	GCS 12/15	6.7	15.2	11.5	34	8.4	124	6.2	64	1.3	2.4k/cumm	2k/cum	16.3	67.4	4.1mmol/l	29	Death
					Not recordable	124/min	102.3	On Mv	Bilateral pupils dilted and fixed	GCS 10/15	6.9	18.4	13.5	56	10.7	120	5.8	58	1.2	4.2k/cumm	9k/cumm	12.5	74.4	4.3mmol/l	34	
Mudishree	3m	Female	681138	Bronchopneumonia , Sepsis in Shock	Not recordable	180/min	97.7	On MV	Bilateral Equal and reactive	GCS 13/15	6.8	13.4	11.5	67	9.7	123	5.7	56	1.9	23k/cumm	134k/cumm	15.2	66.5	4.1mmol/l	20	Recovered
					58/40	179/min	98.6	On MV	Bilateral Equal and reactive	GCS 14/15	6.9	15.6	1.6	108	8.5	107	5.8	45	1.5	21K/CUMM	145k/cumm	16.2	44.4	3.2mmol/l	14	
					68/50	167/min	99.2	On MV	Bilateral Equal and reactive	GCS 15/15	7.1	20.3	16.5	123	12.7	94	5.1	32	1.2	13K/CUMM	224k/cumm	13.4	34.5	2.8mmol/l	8	
B/o Aafiya Sulthana	4m	Female	682516	Bronchopneumonia , Sepsis in Shock	Not recordable	190/min	97/3	On MV	Bilateral Equal and reactive	GCS 10/15	6.8	12.3	12.7	32	10.8	23	6.7	92	2.1	24k/cumm	124k/cumm	21.2	65.4	4.3mmol/l	26	Death
					52/38	188/min	98.4	On MV	Bilateral Equal and reactive	GCS 8/15	6.7	10.8	10.4	60	9.7	120	5.8	78	2.4	18k/cumm	90k/cumm	18.7	60.4	3.8mmol/l	20	
					68/48	179/min	98.3	On MV	Bilateral Equal and reactive	GCS 8/15	6.8	9.2	13.1	57	10.9	98	6.1	80	1.9	17k/cumm	56k/cumm	14.2	67.6	4.6mmol/l	20	
sahana	12 year	Female	682569	Acute GE with severe dehydration	78/50	120/min	99.4	30/min	Bilateral Equal and reactive	GCS 15/15	7.1	23.4	22.8	100	18.5	34	5.4	43	1.2	12k/cumm	123k/cumm	15.6	45.4	3.1mmol/l	12	Recovered
					90/60	124/min	99.6	26/min	Bilateral Equal and reactive	GCS 15/15	7.25	34.5	18.7	96	12.4	98	4.3	32	0.9	13.4k/cumm	245k/cumm	13.2	33.5	2.8mmol/l	5	
					92/60	100/min	99.8	24/min	Bilateral Equal and reactive	GCS 15/15	7.36	38.6	27.8	98	20.5	104	4.3	23	0.6	15k/cumm	260k/cumm	14.3	32.4	1.8mmol/l	3	
Abdul harris	1yr	Male	682551	Acute GE with severe dehydration	Not recordable	168/min	98.7	65/min	Bilateral Equal and reactive	GCS 12/15	6.92	25.3	14.2	100	9.8	105	5.1	34	1.1	12k/cumm	167k/cumm	12.4	46.3	2.8mmol/l	20	Recovered
					70/50	156/min	99.2	56/min	Bilateral Equal and reactive	GCS 13/15	7.16	30.3	24.5	98	18.7	98	5	23	0.9	10k/cumm	150k/cumm	11.5	40.4	2.1mmol/l	12	
					76/57	145/min	99.8	45/min	Bilateral Equal and reactive	GCS 15/15	7.25	32.5	25.4	85	19.1	87	4.2	18	0.6	9.3k/cumm	213k/cumm	12.9	35.4	1.6mmol/l	5	

Name	Age	Sex	IP number	Diagnosis	BP	HR	Temperature	RR	Pupils	GCS	pH	pCO2	Total CO2	PaO2	Bicarbonate	Glucose	Potassium	BUN	Creatinine	WBC	Platelet	PT	PTT	Serum Lactate	PRISM III	Out Come
B/O akir Hussain	7m	Male	682917	Bronchopneumonia , Sepsis in Shock	Not recordable	180/min	97.2	On MV	Bilateral Equal and reactive	GCS 10/15	6.82	12.3	13.4	45	10.8	103	5.7	67	2.1	21K/CUMM	678k/cumm	18.7	68.6	4.2mmol/l	21	Death
					Not recordable	192/min	98.4	On MV	Bilateral pupils dilted and fixed	GCS 8/15	6.9	10.2	11.5	97	7.9	98	6.2	72	1.8	24k/cumm	567k/cumm	17.6	72.5	4.5mmol/l	29	
B/O soundarya	9m	Male	682583	Acute GE with severe dehydration	Not recrdable	167/min	99.4	66/min	Bilateral Equal and reactive	GCS 14/15	7.2	25.3	16.2	102	10.6	86	4.5	35	1.2	12k/cumm	234k/cumm	14.3	49.2	2.8mmol/l	14	Recovered
					65/42	156/min	99.8	54/min	Bilateral Equal and reactive	GCS 15/15	7.32	34.2	20.1	100	13	114	5.2	28	0.9	10.6k/cumm	196k/cumm	13.5	46.3	2mmol/l	6	
					70/50	143/min	99.6	46/min	Bilateral Equal and reactive	GCS 15/15	7.35	36	31.5	90	24.6	102	4.3	20	0.7	11.3k/cumm	245k/cumm	12.4	38.6	1.6mmol/l	3	
B/o Sowmya	2m	Female	683680	Sepsis with septic shock	Not recordable	180/min	98.3	76/min	Bilateral Equal and reactive	GCS 13/15	6.98	13.4	11.9	64	9.8	29	4.3	12	1.3	12k/cumm	347k/cumm	14.3	42.2	3.7mmol/l	15	Recovered
					48/30	176/min	99.6	On MV	Bilateral Equal and reactive	GCS 10/15	6.8	30	16.2	88	10.6	78	4.4	9	0.9	11k/cumm	245k/cumm	15.2	40.7	4mmol/l	11	
					66/46	165/min	99.5	On MV	Bilateral Equal and reactive	GCS 10/15	7.1	35.6	25.7	86	18.2	88	5.1	8	0.7	9k/cumm	256k/cumm	13.6	34.2	3.2mmol/l	1	
B/o Ashwini	2m	Female	683840	Sepsis with septic shock	Not recordable	178/min	99.2	On MV	Bilateral Equal and reactive	GCS 10/15	6.78	13.6	13.4	34	10.7	24	6.2	38	1.6	22.3k/cumm	345k/cumm	12.4	68.4	4.2mmol/l	18	Death
					50/34	167/min	98.6	On MV	Bilateral Equal and reactive	GCS 12/15	6.8	14.2	11.6	80	8.4	108	5.4	46	1.8	20.6k/cumm	460k/cumm	11.8	72.6	4.6mmol/l	14	
					58/38	159/min	100.4	On MV	Bilateral Equal and reactive	GCS 8/15	6.9	15.3	12.7	78	9.8	88	4,8	48	1,5	24k/cumm	540k/cumm	14.2	69	4.1mmol/l	14	
B/o Nethravathi	5m	Male	684769	Bronchopneumonia , Sepsis in Shock	Not recordable	160/min	100.6	88/min	Bilateral Equal and reactive	GCS 10/15	6.92	10.5	13.8	40	10.8	100	5.4	38	0.9	16/cumm	234/cumm	15.3	46.4	3.6mmol/l	18	Recovered
					64/50	164/min	99.7	76/min	Bilateral Equal and reactive	GCS 12/15	7.01	16.3	19.8	68	16.3	96	4.8	27	0.8	23.4k/cumm	231k/cumm	12.4	40.4	3.2mmol/l	8	
					70/54	174/min	98.3	On MV	Bilateral Equal and reactive	GCS 13/15	7.2	20.07	18.6	73	14.2	80	4.3	24	0.5	24.6/cumm	218k/cumm	11.4	36.5	2.8mmol/l	4	
Anushree	11m	Female	685523	Acute GE with severe dehydration	Not recordable	146/min	99.4	67/min	Bilateral Equal and reactive	GCS 15/15	7.12	28	15.7	80	10.8	102	5.2	32	1.2	12.8k/cumm	234k/cumm	14.2	38.7	2.6mmol/l	14	Recovered
					56/40	138/min	99.7	56/min	Bilateral Equal and reactive	GCS 15/15	7.23	32.4	23.2	88	16.2	89	4.6	20	1	10.8k/cumm	378k/cumm	15.3	33.2	2.2mmol/l	14	
					62/50	130/min	98.1	45/min	Bilateral Equal and reactive	GCS 15/15	7.35	40.2	26.8	90	18.4	97	3.9	14	0.6	12k/cumm	217k/cumm	12.4	33.5	1.9mmol/l	10	
B/o Tejashwini	1yr 6 months	Male	687491	Sepsis with septic shock	Not recordable	178/min	99.3	65/min	Bilateral Equal and reactive	GCS 12/15	6.92	24.2	14.5	90	10.6	108	4.1	12	0.7	15k/cumm	235k/cumm	11.4	46.3	3.6mmol/l	13	Recovered
					62/40	168/min	98.3	63/min	Bilateral Equal and reactive	GCS 13/15	7.03	34.2	18.4	88	12.4	92	4.2	10	0.9	12.8k/cumm	216k/cumm	13.2	44.3	3.2mmol/l	5	
					76/56	156/min	99.8	58/min	Bilateral Equal and reactive	GCS 15/15	7.13	46.3	27.3	78	18.2	97	4	18	0.6	11.6k/cumm	216k/cumm	12.4	33.5	2.9mmol/l	2	
Vadhani P	2 year	Female	688107	Sepsis with septic shock	Not recordable	180/min	99.7	68/mn	Bilateral Equal and reactive	GCS 12/15	7.01	28.2	21.8	90	16.2	120	4.5	30	0.9	12.3k/cumm	345k/cumm	15.4	48.7	3.2mmol/l	13	Recovered
					70/52	168/min	99.6	70/min	Bilateral Equal and reactive	GCS 14/15	7.14	30.6	25.3	98	19.5	98	5.1	26	1.1	18k/cumm	213k/cumm	13.2	40.6	2.9mmol/l	10	
					78/60	166/min	98.3	66/min	Bilateral Equal and reactive	GCS 15/15	7.15	35.2	27.8	109	20.3	96	5.3	20	0.7	21K/CUMM	210k/cumm	12.4	33.8	2.3mmol/l	4	
Keshav	1year	Male	689414	Sepsis with septic shock	Not recordable	170/min	99.7	70/min	Bilateral Equal and reactive	GCS 14/15	7.06	23.2	20.6	109	16.2	90	5.1	23	0.9	13.4k/cumm	256k/cumm	13.4	46.7	3.1mmol/l	13	Recovered
					72/44	166/min	98.6	64/min	Bilateral Equal and reactive	GCS 15/15	7.12	32.3	20.5	16.2	14.2	82	5.6	24	0.8	19k/cumm	312k/cumm	12.3	43.3	2.7mmol/l	7	
					76/56	176/min	99.1	60/min	Bilateral Equal and reactive	GCS 15/15	7.34	45.3	27.6	98	18.6	86	4.9	20	0.6	12.4k/cum	360k/cumm	14.3	36.6	2.1mmol/l	4	
Keshavan	2m	Male	689465	Aspiration Pneumonia, Respiratory failure shock	Not recordable	180/min	97.3	ON MV	Bilateral Equal and reactive	GCS 10/15	6.7	12.3	12.8	43	9.8	70	6.1	45	1.2	13.7k/cumm	678k/cumm	15.7	47.3	3.8mmol/l	21	Death
					Not recordable	176/min	98.6	On MV	Bilateral pupils dilted and fixed	GCS 9/15	6.8	9.2	12.6	67	10.5	90	5.8	56	1.4	24k/cumm	614k/cumm	18.2	42.4	4.2mmol/l	32	
B/o Jennifer	4m	Male	689785	Bronchpneumonia Sepsis septic shock	Not recordable	178/min	99.4	67/min	Bilateral Equal and reactive	GCS 12/15	6.9	12.4	10.5	90	8.6	87	6.2	56	1.2	23k/cumm	234k/cumm	12.5	36.4	3.6mmol/l	18	Recovered
					56/40	167/min	99.6	56/min	Bilateral Equal and reactive	GCS 13/15	7.12	13.2	17.6	94	15.2	102	5.8	45	1.1	12.3k/cumm	256k/cumm	11.4	33.4	3.2mmol/l	10	
					66/52	156/min	99.3	52/min	Bilateral Equal and reactive	GCS 14/15	7.2	15.3	21.7	102	18.4	167	4.9	20	0.8	15k/cumm	278k/cumm	13.2	32.1	2.9mmol/l	5	
Sampath	9m	Male	692119	Bronchopneumonia , Sepsis in Shock	Not recordable	168/min	98.3	69/min	Bilateral Equal and reactive	GCS 14/15	7.01	24.2	21.6	87	17.4	91	5.1	18	0.8	11.2k/cumm	245k/cumm	15.2	36.6	3.2mmol/l	12	Recovered
					62/50	156/min	99.6	65/min	Bilateral Equal and reactive	GCS 15/15	7.12	20.7	40.8	92	16.2	95	5.2	12	0.6	12.6k/cumm	345k/cumm	12.3	33.4	2.6mmol/l	5	
					67/48	145/min	99.3	59/min	Bilateral Equal and reactive	GCS 15/15	7.22	28.1	22.8	108	17.6	87	5	14	0.3	13.3k/cumm	213k/cumm	11.6	34.5	2.2mmol/l	2	
B/O Bhagyamma	2m	Male	6912509	Aspiration Pneumonia	Not recordable	190/min	97.3	On MV	Bilateral Equal and reactive	GCS 8/15	6.7	10	11.2	42	8.5	24	5.7	40	1.9	10k/cumm	689k/cumm	15.2	67.3	4.9mmol/l	18	Death
					Not recordable	186/min	99.6	On MV	Bilateral pupils dilted and fixed	GCS 7/15	6.8	9	10.8	56	8.2	60	5.9	42	2.1	11.2k/cumm	700k/cumm	13.4	68	5.0mmol/l	34	
B/o Ashwini	2m	female	692538	Acute GE with severe dehydration	Not recordable	176/min	99.6	66/min	Bilateral Equal and reactive	GCS 15/15	7.12	24	21.5	88	17.4	91	4.5	12	0.7	12k/cumm	167k/cumm	12.2	32.3	3.2mmol/l	9	Recovered
					56/40	167/min	99.2	56/min	Bilateral Equal and reactive	GCS 15/15	7.23	30	22.3	83	16.3	88	4.2	14	0.5	11.6k/cumm	159k/cumm	13.5	33.4	2.6mmol/l	5	
					68/52	156/min	99.1	52/min	Bilateral Equal and reactive	GCS 15/15	7.34	32.4	26.5	100	20.1	102	4.1	10	0.6	10.3k/cumm	178k/cumm	14.3	35.6	2.1mmol/l	0	
Aleena hurin	2years	Female	693781	Acute GE with severe dehydration	Not recordable	160/min	99.2	57/min	Bilateral Equal and reactive	GCS 15/15	6.92	25	11.3	90	5.6	103	5.1	19	0.9	12k/cumm	245k/cumm	15.3	35.6	3.1mmol/l	18	Recovered
					70/54	165/min	99.6	62/min	Bilateral Equal and reactive	GCS 15/15	7.1	24	17.5	94	13.4	107	4.5	12	0.7	12.4k/cum	312k/cumm	14.5	33.8	2.8mmol/l	5	
					80/60	156/min	99.4	66/min	Bilateral Equal and reactive	GCS15/15	7.18	23	20.7	88	16.2	91	4,3	11	0.3	13.8k/cumm	256k/cumm	12.6	32.3	2.4mmol/l	0	
Aravind	17 years	male	693803	Severe Dengue Fever ARDS	Not recordable	140/min	97.2	56/min	Bilateral Equal and reactive	GCS 12/15	6.8	11.3	13.2	34	10.2	68	4.2	24	1	4.3k/cumm	4k/cumm	21.4	46.6	3.9mmol/l	29	Death
					Not recordabel	144/min	98.3	On MV	Bilateral pupils dilted and fixed	GCS 10/15	6.9	10.3	11.7	46	9.4	73	5.2	32	1.2	3.2k/cumm	8k/cumm	19.6	47.5	4.1mmol/l	40	
Dhanushree	3 years	female	69133	Drowning	Not recordable	160/min	97.3	72/min	Bilateral Equal and reactive	GCS 8/15	6.8	9	9.5	28	7.2	29	5.3	30	2.1	26k/cumm	17k/cumm	18.6	49.8	4.2mmol/l	24	Death
					70/50	140/min	98.1	On MV	Bilateral pupils dilted and fixed	GCS 7/15	6.7	10.2	10.4	67	7.8	56	6.1	34	2	23.4k/cumm	25k/cumm	19.5	54.4	4.5mmol/l	42	
Zaid Pasha	2m	Male	696117	Sepsis with septic shock	Not recordable	169/min	99.2	68/min	Bilateral Equal and reactive	GCS 13/15	7.12	12.3	19.8	85	17.8	92	4.3	30	1.8	24k/cumm	678k/cumm	17.8	35.4	3.4mmol/l	14	Recovered

Name	Age	Sex	IP number	Diagnosis	BP	HR	Temperature	RR	Pupils	GCS	pH	pCO2	Total CO2	PaO2	Bicarbonate	Glucose	Potassium	BUN	Creatinine	WBC	Platelet	PT	PTT	Serum Lactate	PRISM III	Out Come
					62/40	172/min	99.5	58/min	Bilateral Equal and reactive	GCS 15/15	7.23	15.6	20.8	90	17.3	114	4.2	26	1.6	23.1k/cumm	656k/cumm	18.3	33.5	3.01mmol/l	10	
					66/42	156/min	99.3	46/min	Bilateral Equal and reactive	GCS 15/15	7.34	24.3	24.5	98	19.2	120	5.1	24	1.4	20k/cumm	567k/cumm	13.4	33.7	2.6mmol/l	7	
B/o Shilpa	3 m	Male	697634	Bronchopneumonia , Sepsis in Shock	Not recordable	170/min	98.3	66/min	Bilateral Equal and reactive	GCS 13/15	7.1	20.3	22.7	90	18.4	140	5.3	30	1.2	13.4k/cumm	345k/cumm	14.3	47.4	3.2mmol/l	14	Recovered
					64/47	166/min	99.3	68/min	Bilateral Equal and reactive	GCS 14/15	7.23	28.6	19.2	98	13.2	120	5.2	22	0.9	12.1k/cum	245k/cumm	12.5	46.6	2.8mmol/l	10	
					66/52	156/min	99.2	63/min	Bilateral Equal and reactive	GCS 15/15	7.3	32.6	24.5	96	18.5	113	4.9	20	0.7	11k/cumm	265k/cumm	11.4	34.5	2.1mmol/l	7	
Dheeraj	1 year	Male	697615	Acute GE with severe dehydration	Not recordable	156/min	98.4	66/min	Bilateral Equal and reactive	GCS 13/15	7.12	29	20.4	90	14.2	87	4.6	26	0.7	19k/cumm	234k/cumm	12.8	32.3	3.2mmol/l	12	Recovered
					66/45	145/min	97.2	64/min	Bilateral Equal and reactive	GCS 15/15	7.23	32.4	23.4	89	17.2	98	4.3	21	0.5	12k/cumm	245k/cumm	13.6	33.6	2.6mmol/l	8	
					72/54	134/min	99.4	63/min	Bilateral Equal and reactive	GCS 15/15	7.34	35.5	27.6	98	21.6	90	4.2	18	0.7	11k/cumm	321k/cumm	11.7	35.6	2.1mmol/l	5	
Prashanth	11yeas	male	698991	Acute GE with severe dehydration	Not recordable	124/min	98.4	34/min	Bilateral Equal and reactive	GCS 14/15	7.12	28.8	26.3	89	20.6	98	5.2	20	1	12k/cumm	234k/cumm	16.7	39.8	3.4mmol/l	13	Recovered
					70/56	143/min	99.3	30/min	Bilateral Equal and reactive	GCS 15/15	7.24	30.9	23.4	92	17.6	78	4.9	22	1.3	12.5k/cumm	304k/cumm	15.3	34.6	3.1mmol/l	10	
					92/60	110/min	99.8	28/min	Bilateral Equal and reactive	GCS 15/15	7.3	32.4	24.6	99	18.4	99	5.1	18	0.9	10.8k/cumm	321k/cumm	13.4	33.6	2.9mmol/l	3	
Abdul Shahadh	3m	male	700318	Bronchopneumonia , Sepsis in Shock	Not recordable	170/min	97.2	On MV	Bilateral Equal and reactive	GCS 12/15	6.8	14.3	12.6	60	10.5	23	6.1	56	1.5	14.8k/cumm	345k/cumm	21.2	42.3	3.9mmol/l	18	Death
					Not Recordable	158/min	97.5	On MV	Bilateral Equal and reactive	GCS 10/15	6.8	18.3	17.2	72	13.2	58	6.5	56	1.4	16k/cumm	356k/cumm	18.6	56.2	4.1mmol/l	18	
					Not recordable	180/min	98.2	On MV	Bilateral pupils dilted and fixed	GCS 10/15	6.78	17.8	13.5	56	9.4	78	5.8	46	1.3	23k/cumm	312k/cumm	22.3	44.6	4.2mmol/l	29	
Fathima Taj	2 years	Female	700761	Acute GE with severe dehydration	Not recordable	170/min	99.7	67/min	Bilateral pupils dilted and fixed	GCS 10/15	7.01	19.7	24.5	90	18.6	87	5.1	23	1.2	12.5k/cumm	319k/cumm	15.4	39.6	2.9mmol/l	14	Recovered
					66/56	158/min	98.6	64/min	Bilateral Equal and reactive	GCS 12/15	7.12	18.5	21.4	98	17.5	88	4.9	20	0.9	13.1k/cumm	345k/cumm	12.3	38.5	2.2mmol/l	10	
					78/60	146/min	98.5	60/min	Bilateral Equal and reactive	GCS 15/15	7.23	25.3	17.6	97	12.6	98	4.2	13	0.8	14.2k/cumm	354k/cumm	11.4	33.4	1.6mmol/l	7	
Thrisha	16 years	Female	701198	Severe Diabeteic Ketoacidosis	Not recordable	120/min	98.3	45/min	Bilateral Equal and reactive	GCS 13/15	6.9	19.6	14.2	92	10.2	HIGH	6.1	12	1.2	28.9k/cumm	235k/cumm	12.3	45.5	3.1mmol/l	17	Recovered
					90/60	109/min	99.2	43/min	Bilateral Equal and reactive	GCS 15/15	7.01	23	20.1	88	16.2	576	5.6	14	0.8	25.8k/cumm	213k/cumm	12.5	43.3	2.8mmol/l	7	
					92/64	108/min	99.7	32/min	Bilateral Equal and reactive	GCS 15/15	7.2	24.1	20.4	102	16.2	456	4.3	18	0.6	21.5k/cumm	245k/cumm	13.2	35.4	2.1mmol/l	7	
Manoj	6 months	Male	701491	Sepsis with septic shock	Not recordable	160/min	99.7	69/min	Bilateral Equal and reactive	GCS 13/15	6.9	14.5	13.1	98	10.6	90	4.5	21	1.2	19.6k/cumm	239k/cumm	13.2	43.3	3.4mmol/l	18	Recovered
					66/45	156/min	100.4	62/min	Bilateral Equal and reactive	GCS 14/15	7.12	18.6	19.5	97	16.2	98	4.2	23	0.9	20.3k/cumm	340k/cumm	11.4	42.2	2.8mmol/l	10	
					78/58	144/min	102.3	56/min	Bilateral Equal and reactive	GCS 15/15	7.23	24.3	21.2	101	17.8	89	5.1	18	0.6	24k/cumm	321k/cumm	12.6	35.6	2.3mmol/l	4	
Mahira	2 years	Female	702075	Sepsis with septic shock	Not recordable	170/min	98.3	On MV	Bilateral Equal and reactive	GCS10/15	6.7	12.3	11.5	26	9	90	6.1	23	1.2	26.6k/cumm	321k/cumm	23.8	60.4	3.9mmol/l	18	Death
					Not recordable	180/min	97.3	On MV	Bilateral Equal and reactive	GCS 10/15	6.8	18.3	15.8	56	12.4	90	6.3	26	1	25.3k/cumm	236k/cumm	25.3	65.6	4.2mmol/l	18	
					Not recordable	178/min	99.5	On MV	Bilateral pupils dilted and fixed	GCS 9/15	6.8	10.8	15.6	78	13.2	92	5.2	20	0.9	25k/cumm	370k/cumm	14.5	53.3	4.3mmol/l	29	
B/o Nooruzuha	6years	Male	671511	Acute GE with severe dehydration	Not recordable	168/min	99.4	66/min	Bilateral pupils dilted and fixed	GCS 12/15	7.1	12.3	13.8	80	11.1	90	5.2	23	1.2	23k/cumm	324k/cumm	12.4	46.4	3.4mmol/l	14	Recovered
					88/56	170/min	99.2	64/min	Bilateral Equal and reactive	GCS 14/15	7.23	14.3	15.7	92	13.6	108	5.6	21	1.1	25.1k/cumm	321k/cumm	11.2	41.1	3,1mmol/l	10	
					92/60	182/min	98.2	62/min	Bilateral Equal and reactive	GCS 15/15	7.32	20.4	28.7	102	24.2	100	4.8	18	0.9	18.3k/cumm	345k/cumm	13.2	36.4	2.9mmol/l	5	
Akshitha	4 years	Female	703911	Severe Diabeteic Ketoacidosis	Not recordable	154/min	98.3	54/min	Bilateral Equal and reactive	GCS 15/15	7.12	20.1	16.8	100	12.4	HIGH	5.2	21	1.4	21k/cumm	231k/cumm	11.5	47.6	3.2mmol/l	16	Recovered
					84/60	135/min	99.4	50/min	Bilateral Equal and reactive	GCS 15/15	7.23	21.1	18.2	107	13.4	467	5.1	22	1.2	23.4k/cumm	213k/cumm	12.5	42.2	3.1mmol/l	13	
					88/64	120/min	99.6	46/min	Bilateral Equal and reactive	GCS 15/15	7.34	24	26.5	98	21.5	450	5.4	23	0.9	12.7k/cumm	245k/cumm	13.2	38.4	2.5mmol/l	8	
Anup Gowda	3 years Male	Male	703966	Bronchpneumonia Sepsis septic shock	Not recordable	166/min	98.3	78/min	Bilateral Equal and reactive	GCS 14/15	6.9	15	12.3	56	8.7	96	6.1	22	0.9	12.4k/cum	256k/cumm	12.7	47.5	3.7mmol/l	18	Recovered
					72/50	156/min	99.3	On MV	Bilateral Equal and reactive	GCS 15/15	7.01	23	17.5	97	13.2	92	5.6	23	1.1	16.2k/cumm	245k/cumm	13.6	43.3	3.01mmol/l	10	
					76/54	146/min	99.2	On MV	Bilateral Equal and reactive	GCS 15/15	7.23	21.4	20.5	100	16.2	90	5.2	25	1.2	17k/cumm	312k/cumm	13.5	36.6	2.9mmol/l	10	
Anish	1year 5 months	Male	704410	Bronchopneumonia , Sepsis in Shock	Not recordable	170/min	98.3	78/min	Bilateral Equal and reactive	GCS 12/15	6.9	21	13.2	109	8.6	103	4.8	26	1.1	21k/cumm	345k/cumm	11.9	40.4	3.6mmol/l	18	Recovered
					66/45	167/min	98.6	76/min	Bilateral Equal and reactive	GCS 13/15	7.01	20.6	16.1	96	12.3	110	5.2	23	0.9	16.6k/cumm	321k/cumm	15	36.5	3.1mmol/l	10	
					78/60	156/min	99.4	75/min	Bilateral Equal and reactive	GCS 12/15	7.12	24	23.1	97	17.3	123	5.1	21	0.8	12k/cumm	290k/cumm	12.7	33.4	2.8mmol/l	4	
Jeevan	1 year	Male	705124	Acute GE with severe dehydration	Not recordable	156/min	99.8	68/min	Bilateral Equal and reactive	GCS 13/15	7.02	20	22.6	98	18.5	67	4.3	21	0.8	21.3k/cumm	256k/cumm	13.2	46.7	2.8mmol/l	12	Recovered
					64/45	145/min	99.3	56/min	Bilateral Equal and reactive	GCS 14/15	7.14	23	17.6	96	13.2	89	4,1	12	0.7	13.4k/cumm	341k/cumm	14.3	42	2.2mmol/l	5	
					78/60	134/min	99.4	45/min	Bilateral Equal and reactive	GCS 15/15	7.24	30.3	21.3	92	15.6	92	3.9	10	0.6	14.2k/cumm	289k/cumm	12.5	36	1.8mmol/l	2	
B/O Bharathi	5m	Male	706489	Acute GE with severe dehydration	Not recordable	169/min	99.2	56/min	Bilateral Equal and reactive	GCS 14/15	7.01	22	23.5	108	19.1	23	4.2	28	1.2	12k/cumm	234k/cumm	13.6	47.5	3.1mmol/l	14	Recovered
					64/46	156/min	99.4	46/min	Bilateral Equal and reactive	GCS 15/15	7.2	20	23.4	80	19.2	67	5.1	22	0.9	18k/cumm	213k/cumm	12.4	42.2	2.7mmol/l	10	
					66/54	146/min	98.2	44/min	Bilateral Equal and reactive	GCS 15/15	7.32	24.3	21.5	90	17.2	88	4.9	18	0.8	21K/CUMM	245k/cumm	13.2	38.3	2.1mmol/l	3	
Infant	11m	Male	707022	Bronchpneumonia Sepsis septic shock	Not recordable	160/min	99.1	76/min	Bilateral Equal and reactive	GCS 12/15	7.01	21.1	15.3	98	10.6	45	4.2	12	0.9	23.4k/cumm	213k/cumm	13.8	46	3.4mmol/l	9	Recovered
					66/54	168/min	99.6	68/min	Bilateral Equal and reactive	GCS 13/15	7.21	23.4	16.5	101	12.5	87	3.8	10	0.7	22.1k/cumm	223k/cumm	12.5	42	3.1mmol/l	5	
					78/56	158/min	99.4	72/min	Bilateral Equal and reactive	GCS15/15	7.28	25.1	25.8	87	19.2	90	4.6	12	0.6	20.4k/cumm	167k/cumm	11.5	33.4	2.8mmol/l	3	

Name	Age	Sex	IP number	Diagnosis	BP	HR	Temperature	RR	Pupils	GCS	pH	pCO2	Total CO2	PaO2	Bicarbonate	Glucose	Potassium	BUN	Creatinine	WBC	Platelet	PT	PTT	Serum Lactate	PRISM III	Out Come
Merisha	1 yr	Feamle	707843	Bronchopneumonia , Sepsis in Shock	Not recordable	190/min	96.2	On MV	Bilateral Equal and reactive	GCS 8/15	6.7	10	11.2	23	9.2	45	5.6	34	1.9	21K/CUMM	678k/cumm	16.4	46.5	3.8mmol/l	18	Death
					Not recordable	186/min	99.6	On MV	Bilateral pupils dilted and fixed	GCS 9/15	6.8	9	11.6	68	8.6	98	5.2	45	1.7	22.3k/cumm	768k/cumm	12.7	56.6	4.1mmol/l	29	
Tharun Kumar	17 year	Male	708440	Severe Dengue Fever	Not recordable	136/min	98.3	34/min	Bilateral pupils dilted and fixed	GCS 15/15	7.12	28	16.5	98	11.6	90	5.2	10	0.8	4k/cumm	34k/cumm	13.2	36.6	3.1mmol/l	15	Recovered
					88/60	142/min	99.3	23/min	Bilateral Equal and reactive	GCS 15/15	7.32	32.5	36.2	108	20.3	89	5.1	12	0.6	3.5k/cumm	23k/cumm	12.4	33.4	2.8mmol/l	6	
					92/70	120/min	100.2	24/min	Bilateral Equal and reactive	GCS 15/15	7.4	38.3	31.2	124	26.3	78	4.9	19	0.5	8k/cumm	12k/cumm	12.6	31.1.	2.1mmol/l	6	
B/o Anuradha	3months	female	709496	Bronchopneumonia, Sepsis in shock	Not recordable	180/min	98.4	74/min	Bilateral Equal and reactive	GCS 13/15	7.01	12.4	18.3	80	16.3	80	4.4	12	0.9	16k/cumm	254k/cumm	11.4	47.5	3.6mmol/l	9	Recovered
					56/40	172/min	98.2	68/min	Bilateral Equal and reactive	GCS 14/15	7.12	15.6	23.5	93	20.3	98	4.6	18	1.1	12k/cumm	212k/cumm	12.4	38.6	2.8mmol/l	7	
					68/46	164/min	99.2	56/min	Bilateral Equal and reactive	GCS 15/15	7.23	20	25.3	100	21.2	101	4.2	16	0.7	11.3k/cumm	318k/cumm	13.2	33.3	2.1mmol/l	7	
Vijayalakshmi	16yrs	Female	391852	Severe Dengue Fever	Not recordable	120/min	98.2	38/min	Bilateral Equal and reactive	GCS 15/15	7.02	13.4	29.3	98	12.6	78	3.8	12	0.8	11k/cumm	5k/cumm	14.2	56	2.8mmol/l	15	Recovered
					76/50	106/min	99.2	34/min	Bilateral Equal and reactive	GCS 15/15	7.13	20.4	40.3	92	16.2	98	4.5	11	0.6	12k/cumm	12k/cumm	15.6	43	2.1mmol/l	11	
					90/62	92/min	100.1	32/min	Bilateral Equal and reactive	GCS 15/15	7.24	25.4	21.6	100	16.3	102	4.2	10	0.7	13k/cumm	30k/cumm	12.4	37	1.5mmol/l	11	
Florence	7 years	Female	710860	Acute GE with severe dehydration	Not recordable	120/min	99.7	38/min	Bilateral Equal and reactive	GCS 13/15	7.12	26	17.6	102	12.6	86	4.3	12	1.1	12k/cumm	234k/cumm	13.2	49.5	2.6mmol/l	11	Recovered
					78/50	113/min	98.2	36/min	Bilateral Equal and reactive	GCS 15/15	7.23	28	21.4	98	16.3	98	4.2	13	0.9	15k/cumm	218k/cumm	11.4	46.5	2.2mmol/l	11	
					84/60	110/min	100.3	28/min	Bilateral Equal and reactive	GCS 15/15	7.3	30	23.1	92	17.6	100	4.8	10	0.8	16k/cumm	267k/cumm	13.2	43.3	1.6mmol/l	9	
Arun	1year 2 months	Male	713021	Dengue Fever with warning signs	Not recordable	140/min	98.3	42/min	Bilateral Equal and reactive	GCS 14/15	7.12	28.3	17.6	98	12.3	88	4.1	12	0.8	12.3k/cumm	4k/cumm	16.4	36.5	3.1mmol/l	15	Recovered
					72/45	125/min	99.2	38/min	Bilateral Equal and reactive	GCS 15/15	7.23	30.1	27.3	96	21.3	100	4.5	13	0.7	13.2k/cumm	8k/cumm	12.3	32.2	2.5mmol/l	11	
					78/56	112/min	100.01	36/min	Bilateral Equal and reactive	GCS 15/15	7.35	34.2	32.1	100	26.1	102	4.6	14	0.7	4k/cumm	10k/cumm	14.3	33.4	1.6mmol/l	6	
Punith Raj	12 years	Female	714084	OP Compound Poisoning	Not Recordable	160/min	97.3	43/min	Bilateral Equal and reactive	GCS 8/15	6.8	10.4	11.6	76	9.5	24	4.3	43	2.1	34k/cumm	345k/cumm	12.2	46.6	4.1mmol/l	28	Death
					Not recordable	154/min	98.3	On MV	Bilateral pupils dilted and fixed	GCS 7/15	6.9	12.3	10.6	65	8.5	90	4.6	56	1.9	24k/cumm	213k/cumm	13.2	56.5	3.8mmol/l	34	
B/O Bharathi	4m	Male	714068	Acute GE with severe dehydration	Not recordable	140/min	98.4	On MV	Bilateral Equal and reactive	GCS 8/15	6.8	19	13.2	100	9.6	98	4.3	46	2.1	21K/CUMM	256k/cumm	12.1	49.8	4.1mmol/l	18	Death
					Not recordable	154/min	99.2	On MV	Bilateral pupils dilted and fixed	GCS 7/15	6.9	21	14.6	98	10.3	87	5.4	48	2.3	26k/cumm	234k/cumm	11.3	56	4.6mmol/l	34	
Vishwas	2m	Male	714378	Severe Sepsis in Shock	Not recordable	160/min	98.3	78/min	Bilateral Equal and reactive	GCS 10/15	6.9	9.4	12.1	68	9.8	98	4	28	1.2	24k/cumm	213k/cumm	13.2	48.8	3.8mmol/l	18	Recovered
					66/57	146/min	99.2	72/min	Bilateral Equal and reactive	GCS 12/15	7.01	10.7	15.3	98	13.2	92	5.4	26	1	18k/cumm	276k/cumm	11.4	38.6	3.2mmol/l	9	
					78/66	136/min	100.3	70/min	Bilateral Equal and reactive	GCS 13/15	7.12	12.4	17.1	102	14.7	101	4.2	24	0.8	14k/cumm	312k/cumm	13.2	37.7	2.8mmol/l	5	
Syed Zunaid	3m	Male	714098	Aspiration Pneumonia	Not recordable	180/min	99.7	On MV	Bilateral Equal and reactive	GCS 9/15	6.8	15.5	13.2	45	10.2	28	4.3	28	1.9	34k/cumm	230k/cumm	15.3	59.7	4.2mmol/l	21	Death
					Not recordable	198/min	98.6	On MV	Bilateral pupils dilted and fixed	GCS 8/15	6.9	18.2	12.6	98	9.4	93	4.6	21	1.6	26k/cumm	320k/cumm	14.2	47.8	3.8mmol/l	32	
B/o Sathyaveni	2m	Female	716371	Sepsis with septic shock	Not recordable	180/min	98.3	76/min	Bilateral pupils dilted and fixed	GCS 10/15	6.8	13.4	11.3	100	10.6	28	5.2	34	2.2	23.3k/cumm	321k/cumm	12.6	47	3.4mmol/l	14	Recovered
					58/46	176/min	99.4	68/min	Bilateral Equal and reactive	GCS 12/15	7.01	25.3	21.3	102	16.5	98	4.8	32	1.8	22.3k/cumm	256k/cumm	12.4	36.6	3.2mmol/l	10	
					68/50	169/min	100	64/min	Bilateral Equal and reactive	GCS 13/15	7.2	26	23.4	98	18.2	100	4.3	30	1.7	20k/cumm	302k/umm	12.3	33.4	2.9mmol/l	10	
Kubra	8m	female	718290	Acute GE with severe dehydration	Not recordable	180/min	98.2	70/min	Bilateral Equal and reactive	GCS 12/15	7.02	23	15.1	90	11.6	108	4.5	32	2.1	21k/cumm	367k/cumm	11.5	46.6	3.8mmol/l	14	Recovered
					62/50	176/min	99	68/min	Bilateral Equal and reactive	GCS 14/15	7.12	32	21.5	89	15.6	123	4.2	26	1.9	13.5k/cumm	234k/cumm	15.3	43.3	3.2mmol/l	10	
					78/56	168/min	100	62/min	Bilateral Equal and reactive	GCS 15/15	7.2	36.6	19.2	88	12.4	112	4.8	28	1.7	12k/cumm	212k/cumm	12.4	38.6	2.8mmol/l	7	
Vishwas	3m	Male	719295	Aspiration Pneumonia	Not Recordable	188/min	99.2	78/min	Bilateral Equal and reactive	GCS 10/15	6.9	15.7	14.6	45	9.4	45	4.5	12	0.9	16k/cumm	220k/cumm	12.6	49.5	3.4mmol/l	13	Recovered
					54/42	192/min	99	76/min	Bilateral Equal and reactive	GCS 12/15	7.06	25.2	18.6	65	13.5	88	4.7	14	0.8	14k/cumm	345k/cumm	12.8	45.5	2.9mmol/l	7	
					60/48	176/min	99.8	On MV	Bilateral Equal and reactive	GCS 12/15	7.12	30.5	19.5	101	13.2	123	4.2	15	1.1	12.3k/cumm	230k/cumm	11.6	33.2	2.1mmol/l	7	
Kushal Gowda	2m	Male	719389	Sepsis with septic shock	Not recordable	188/min	96.3	On MV	Bilateral Equal and reactive	GCS 7/15	6.8	20.6	13.4	45	9.4	40	6.1	45	2.1	4k/cumm	15k/cumm	12.4	45.5	4.2mmol/l	30	Death
					Not recordable	198/min	99.6	On MV	Bilateral pupils dilted and fixed	GCS 8/15	6.9	23.5	13.6	68	8.6	123	6.7	60	2.2	12k/cumm	25k/cumm	13.2	50.3	4.5mmol/l	35	
B/O Arshiya Siddiqui	2m	Female	726639	Sepsis with Septic Shock	Not recordable	180/min	96.5	78/min	Bilateral Equal and reactive	GCS 10/15	6.8	9.7	11.8	88	9.6	103	5.4	67	2.3	2.9k/cumm	678k/cumm	11.7	46.6	3.6mmol/l	18	Recovered
					56/40	192/min	97.3	On MV	Bilateral Equal and reactive	Gcs 8/15	7.01	10.2	15.4	56	13.8	112	4.6	72	1.8	3k/cumm	767k/cumm	12.4	38.8	3.4mmol/l	12	
					68/56	176/min	99	On MV	Bilateral Equal and reactive	GCS 8/15	7.11	18.3	21.5	76	18.2	123	4.8	112	1.6	4.2k/cumm	564k/cumm	13.2	33.3	3.3mmol/l	9	
Manasa	8y	Female	727346	Dengue Fever with warning signs	Not recordable	124/min	96.3	34/min	Bilateral Equal and reactive	GCS 15/15	7.1	20	16.5	120	12.3	100	4.2	12	0.7	4.5k/cumm	12k/cumm	14.2	34.3	2.8mmol/l	15	Recovered
					90/50	136/min	97.2	32/min	Bilateral Equal and reactive	GCS 15/15	7.25	28	20.3	112	14.6	97	4.1	13	0.6	12k/cumm	23k/cumm	12.5	33.3	2.1mmol/l	8	
					96/60	110/min	99.3	36/min	Bilateral Equal and reactive	GCS 15/15	7.32	35.3	23.4	143	16.2	97	3.6	12	0.5	11k/cumm	10k/cumm	13.2	32.2	1.4mmol/l	6	
Poojashree	12 yrs	Female	728087	Dengue Fever with warning signs	Not recordable	112/min	100.2	28/min	Bilateral Equal and reactive	GCS 15/15	7.2	35.5	20.3	108	13.4	80	3.7	15	0.8	12k/cumm	10k/cumm	12.6	36.6	2.7mmol/l	15	Recovered
					90/55	120/min	101.4	32/min	Bilateral Equal and reactive	GCS 15/15	7.32	38.2	23.3	97	16.2	87	4.1	13	0.7	12.3k/cumm	15k/cumm	11.8	33.4	2.1mmol/l	6	
					92/60	98/min	99.6	24/min	Bilateral Equal and reactive	GCS 15/15	7.4	40	28.1	90	20.1	90	4,5	10	0.3	11k/cumm	4k/cumm	12.3	32.3	1.3mmol/l	6	
Manvitha	2m	Female	728185	Bronchopneumonia with sepsis in septic shock	Not recordable	178/min	99.8	66/min	Bilateral Equal and reactive	GCS 14/15	7.03	34	18.6	134	12.3	98	4.2	12	0.8	12k/cumm	234k/cumm	15.5	43.3	4.2mmol/l	9	Death

Name	Age	Sex	IP number	Diagnosis	BP	HR	Temperature	RR	Pupils	GCS	pH	pCO2	Total CO2	PaO2	Bicarbonate	Glucose	Potassium	BUN	Creatinine	WBC	Platelet	PT	PTT	Serum Lactate	PRISM III	Out Come
					52/43	165/min	99.6	56/min	Bilateral Equal and reactive	GCS 15/15	7.12	40	24.6	154	16.3	87	4.1	16	0.9	14k/cumm	290k/cumm	17.2	38	3.9mmol/l	14	
					60/44	177/min	99	54/min	Bilateral Equal and reactive	GCS 15/15	7.4	52	28.4	112	18.4	90	4.4	20	1	28k/cumm	312k/cumm	13.4	33	4.6mmol/l	14	
Sandhya	16 yrs	Female	728594	Dengue Fever with warning signs	Not recordable	130/min	98.3	34/min	Bilateral Equal and reactive	GCS 15/15	7.2	34	24.6	157	16.4	123	4.2	12	0.5	2k/cumm	23k/cumm	12.6	46	2.7mmol/l	19	Recovered
					90/54	128/min	99.6	28/min	Bilateral Equal and reactive	GCS 15/15	7.32	38	31.6	120	23.4	112	3.7	12	0.6	2.9k/cumm	10k/cumm	11.8	43	2.1mmol/l	10	
					94/60	112/min	100.8	24/min	Bilateral Equal and reactive	GCS 15/15	7.4	45.2	35.3	98	26.3	132	3.3	13	0.7	4.7k/cumm	18k/cumm	12.3	38.6	1.8mmol/l	6	
Hemanth Reddy	17y	Male	728799	Dengue Fever with warning signs	Not recordable	120/min	100.2	28/min	Bilateral Equal and reactive	GCS15/15	7.12	35.4	27.3	90	20.6	111	3.5	12	0.7	4k/cumm	12k/cumm	14.2	45.5	2.6mmol/l	15	Recovered
					90/60	112/min	99.8	24/min	Bilateral Equal and reactive	GCS 15/15	7.2	33.3	24.6	88	18.2	132	4.1	16	0.6	5.6k/cumm	10k/cumm	13.2	43.3	2.2mmol/l	8	
					98/65	100/min	99.3	22/min	Bilateral Equal and reactive	GCS 15/15	7.35	36	33.5	100	26.6	100	4.6	17	0.3	6.9k/cumm	45k/cumm	12.5	38.6	1.2mmol/l	6	
Swathi	14 years	Female	729884	Severe Dengue Fever	Not recordable	120/min	99.6	28/min	Bilateral Equal and reactive	GCS 15/15	7.23	28.6	30.3	119	24.3	127	4.2	12	0.8	7.2k/cumm	12k/cumm	15.3	46	3.6mmol/l	15	Recovered
					90/50	132/min	102.2	24/min	Bilateral Equal and reactive	GCS 15/15	7.34	37.2	33.6	123	26.2	76	4.5	12	0.6	6,8k/cumm	15k/umm	13.2	34.4	2.8mmol/l	6	
					98/60	128/min	100.4	20/min	Bilateral Equal and reactive	GCS 15/15	7.4	42.6	36.2	145	28.2	99	4.2	14	0.5	12k/cumm	12k/cumm	14.3	32.1	1.7mmol/l	6	
B/o Laksmi devi	2 months	Male	730981	Severe Sepsis in Shock	Not recordable	180/min	101.2	78/min	Bilateral Equal and reactive	GCS 10/15	6.8	21	14.6	120	10.2	90	4.3	16	0.6	26k/cumm	678k/cumm	16.3	65.6	4.6mmol/l	16	Death
					Not recordable	172/min	99.3	On MV	Bilateral pupils dilted and fixed	GCS 8/15	6.7	23	16.5	124	12.3	92	4.6	19	0.8	12k/cumm	564k/cumm	13.4	72.3	5.01mmol/l	27	
Rakshay	10 years	Male	730984	Dengue Fever with warning signs	Not recordable	98/min	98.6	26/min	Bilateral Equal and reactive	GCS 15/15	7.1	26	22.3	89	16.2	102	4.6	12	0.6	13k/cumm	15k/cumm	12.2	33.6	2.6mmol/l	15	Recovered
					90/60	92/min	99.2	22/min	Bilateral Equal and reactive	GCS 15/15	7.2	24.3	23.2	96	18.6	130	4.8	16	0.8	14k/cumm	12k/cumm	11.2	34.3	2.1mmol/l	8	
					96/60	84/min	99.2	24/min	Bilateral Equal and reactive	GCS 15/15	7.32	30.1	34.3	102	20.3	121	4.1	13	0.6	21k/cumm	13k/cumm	14.3	35.3	1.6mmol/l	8	
Rakesh	11years	Male	730965	Dengue Fever with warning signs	Not recordable	82/min	98.6	26/min	Bilateral Equal and reactive	GCS 15/15	7.12	24	32.3	92	20.4	120	4.3	12	0.5	25k/cumm	12k/cumm	12.3	36.3	2.2mmol/l	15	Recovered
					90/58	98/min	99.2	24/min	Bilateral Equal and reactive	GCS 15/15	7.2	26.3	34.3	97	22.6	114	4.2	14	0.4	19k/cumm	21k/cumm	14.3	34.3	1.9mmol/l	8	
					98/60	100/min	98.6	28/min	Bilateral Equal and reactive	GCS 15/15	7.24	28	35.3	100	24.3	98	4.1	13	0.3	15k/cumm	13k/cumm	14	38.3	1.4mmol/l	8	
Kavya	12 years	Female	730990	Dengue Fever with warning signs	Not recordable	92/min	98.6	26/min	Bilateral Equal and reactive	GCS 15/15	7.04	24	26.2	86	22.3	92	4.3	13	0.4	13.2k/cumm	120k/cumm	12.3	28.3	2.5mmol/l	15	Recovered
					92/60	106/min	99.4	21/min	Bilateral Equal and reactive	GCS 15/15	7.24	28	29.3	92	24.6	96	4.1	14	0.8	15k/cumm	98k/cumm	13.4	33.2	1.8mmol/l	8	
					96/70	100/min	97.6	24/min	Bilateral Equal and reactive	GCS 15/15	7.32	30	32.3	100	26.3	102	4.8	12	0.7	12.7k/cumm	90k/cumm	13	34.3	1.2mmol/l	8	
Sai Ram	15 years	Male	730996	Dengue Fever with warning signs	Not recordable	86/min	98.6	28/min	Bilateral Equal and reactive	GCS 15/15	7.12	26	25.3	98	20.3	86	4.3	13	0.8	15k/cumm	15k	12.6	35.3	2.3mmol/l	15	Recovered
					92/60	90/min	99.3	27/min	Bilateral Equal and reactive	GCS 15/15	7.26	28.3	30.2	96	24.3	102	4.2	10	0.7	13k/cumm	10k/cumm	14.3	34.3	2.1mmol/l	8	
					100/60	92/min	97.3	25/min	Bilateral Equal and reactive	GCS 15/15	7.42	32.4	34.2	100	26.2	106	4.1	13	0.9	12.5k/cumm	9k/cumm	12.4	33.6	1.6mmol/l	8	
Manasa	12 years	Female	730998	Dengue Fever with warning signs	Not recordable	100/min	97.3	24/min	Bilateral Equal and reactive	GCS 15/15	7.06	23.3	26.3	88	20.2	92	5.2	10	0.6	11.6k/cumm	21k/cumm	13.2	33.5	2.7mmol/l	15	Recovered
					84/56	95/min	98.3	20/min	Bilateral Equal and reactive	GCS 15/15	7.16	26.3	27.2	92	22.3	86	4.6	12	0.4	13.2k/cumm	26k/cumm	14.2	33.4	2.2mmol/l	8	
					96/60	96/min	99.3	26/min	Bilateral Equal and reactive	GCS 15/15	7.23	28.3	30.1	97	24.2	90	3.8	11	0.7	15.6k/cumm	120k/cumm	13.2	32.2	1.8mmol/l	5	