

**KLE ACADEMY OF HIGHER EDUCATION AND RESEARCH,
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**“PREVALENCE AND CLINICAL OUTCOME OF HYPERGLYCAEMIA
IN CRITICALLY ILL CHILDREN AGED 2 MONTHS TO 18 YEARS,
A ONE YEAR CROSS-SECTIONAL STUDY”.**

BY

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IN
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**DEPARTMENT OF PEDIATRICS,
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BELAGAVI, KARNATAKA**

i

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LIST OF ABBREVIATIONS USED

Acronym	Full Form
ICU	Intensive Care Unit
TNF	Tumor Necrosis Factor
IL	Interleukin
HPA	Hypothalamic Pituitary Adrenal Axis
BCE	Before Common Era
BC	Before Christ
PICU	Paediatric Intensive Care Unit
PAT	Paediatric Assessment Triangle
PALS	Paediatric Advanced Life Support
ECF	Etra-cellular Fluid
ICF	Intra-cellular Fluid
POSM	Plasma Osmolality

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Acronym	Full Form
PNa	Plasma Sodium
RBC	Red Blood Cells
ACTH	Adrenocorticotropic Hormone
DNA	Deoxyribonucleic Acid
GCS	Glasgow Coma Scale
WHO	World Health Organization
SD	Standard Deviation
QQ	Quantile Quantile Plot
MIN	Minimum
MC	Monte Carlo Simulation
CNS	Central Nervous System
GIT	Gastrointestinal Tract
C	Chi Square Test
RAAS	Renin Angiotensin Aldosterone System
VLDL	Very Low Density Lipoprotein

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LDL	Low Density Lipoprotein
HDL	High Density Lipoprotein
GLUT	Glucose Transporter
FFA	Free Fatty Acids
NICU	Neonatal Intensive Care Unit
PRISM	Paediatric Risk Of Mortality Score
TPN	Total Parenteral Nutrition
MSS	Meningococcal Septic Shock
ABG	Arterial Blood Gas
PT	Prothrombin Time
APTT	Activated Partial Thromboplastin Time
CBC	Complete Blood Count
ANOVA	Analysis Of Variance
MW	Mann Whitney U test

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ABSTRACT

“PREVALENCE AND CLINICAL OUTCOME OF HYPERGLYCAEMIA IN CRITICALLY ILL CHILDREN- A ONE YEAR CROSS SECTIONAL STUDY AT KLE’S DR PRABHAKAR KORE HOSPITAL, BELGAUM”.

INTRODUCTION:

In normal healthy state, human body maintains homeostasis and normal glycemic levels. However, in stress associated with any critical states, this control is impaired or lost. Hyperglycemia represents extreme form of stress.

OBJECTIVES:

Primary objective- To find out the prevalence of hyperglycemia in critically ill children admitted in PICU.

Secondary objective- assess the clinical outcomes among hyperglycemic based on PICU stay, inotropes used, mechanical ventilation and final outcome i.e., discharged healthy or death.

METHODS:

A one-year cross sectional study, from January 2021 to Dec 2021, at a tertiary care center. 284 patients between the ages of 1month and 18 years admitted in PICU who were critically ill were enrolled in the study, by considering inclusion and exclusion criteria. Patient were followed throughout the duration of PICU stay and outcome. In all cases blood glucose estimation was done in laboratory by hexokinase method. Glucose estimation was done first at the admission

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then every 12th hourly in all cases. If the child falls under hyperglycemia (>126 mg/dl) the child will be monitored every 6th hourly till the glucose level falls below 126mg/dl. Other lab investigations that are ABG, PT, APTT, serum bilirubin, potassium, calcium, complete blood count which were required for PRISM III score were done. PRISM III was done initially after admission and after 4 days in PICU to predict mortality and severity and correlate with blood glucose levels.

RESULTS:

Among 284 children included in the study group from 1 month to 18 years, 86 (30.28%) were infants, 131 (46.13%) in the age group of 1-10 years, 67 (23.59%) were more than 10 years. 119 (41.9%) were female and 165 (58.1%) were males. Among these 284 patients, 2 children (0.7%) had a blood glucose level of <40 mg/dl, 163 children (57.39%) had an admission blood glucose of 41-125 mg/dl and 119 children (41.9%) had blood glucose level of >126 mg/dl. There was a statistically significant association between hyperglycemia and those cases requiring mechanical ventilator, inotropes, and PICU stay. PRISM III score was higher in those with hyperglycaemias. Outcome was poor in those cases which had hyperglycaemia and risk of death was more in them.

CONCLUSION:

Prevalence of mean hyperglycaemia was 31.93%, 25.21%, 42.86% when cut off levels of blood glucose was considered as >126 mg/dl, >150 mg/dl, >200 mg/dl respectively. There was an increased risk of mortality and morbidity in critically ill children having hyperglycaemia.

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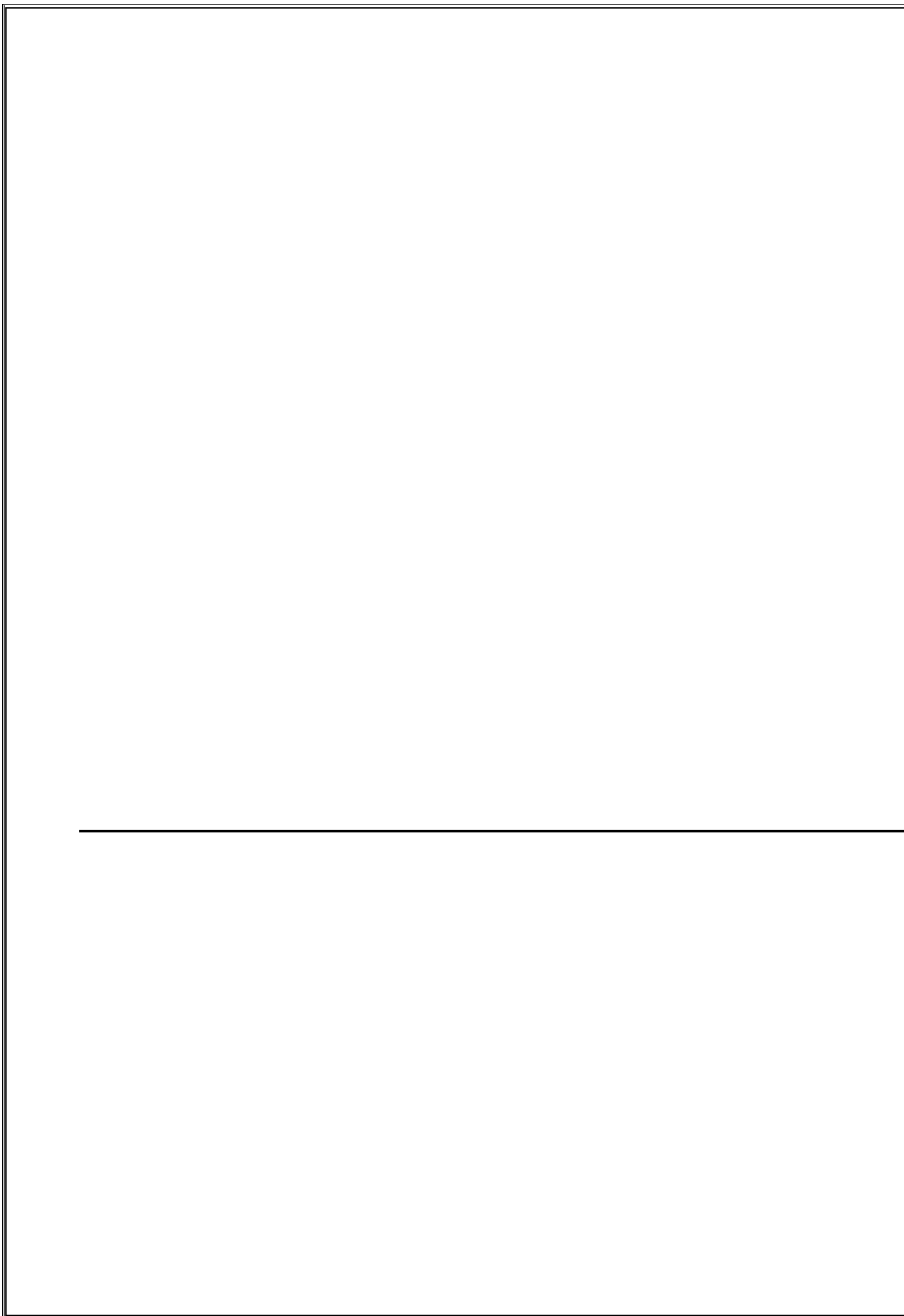
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INTRODUCTION

INTRODUCTION

HYPERGLYCEMIA:

The term hyperglycaemia, is originated in Greek. Where hyper meaning excessive, glyc meaning sweet and emia meaning in the blood. Hence hyperglycaemia is a condition with excessive amount of glucose circulating in the blood plasma. Hyperglycaemia is defined as blood glucose >126 mg/dl (1, 2)

STRESS HYPERGLYCEMIA:

Stress hyperglycaemia is also called as stress diabetes. Human body maintains normal glycaemic levels independent of food and energy expenditure, in a normal healthy state. Whereas in stress associated with any critical illness this control gets impaired, leading to hyperglycaemia Stress hyperglycaemia refers to the transient increase in the blood plasma glucose during stress of an illness.it is seen in patients who has no previous evidence or history of diabetes.

It often gets discovered when the routine blood glucose measurements are done in a sick child admitted in the hospital.

Blood glucose can be monitored either by the plasma glucose test performed in a laboratory or by a bedside test by using glucometer. Though the laboratory test is more reliable and more efficacious.

Stress hyperglycaemia usually occurs during the critical illness in children even with previously normal glucose homeostasis (3-7). Stress hyperglycaemia can be prolonged for days

to weeks when the child is admitted in ICU. Non-survivors have high blood glucose concentration and also tend to have longer duration of stress hyperglycaemia as compared to survivors.

Hyperglycaemia in critically ill children results from increased release of counter regulatory hormones, peripheral insulin resistance, and oxidative stress and certain therapeutic interventions. Glucocorticoid and adrenaline promote lipolysis and further cause insulin resistance in skeletal muscle. Cytokines also play role in stress hyperglycaemia. $\text{TNF}\alpha$, IL-1, IL-6 stimulate 3 HPA axis leading to increased glucocorticoid levels in the blood. $\text{TNF}\alpha$ also induces insulin resistance directly.

Initially stress hyperglycaemia during critical illness was thought to be an adaptive response but no longer considered as a physiological response as it's associated with increased morbidity and mortality.

Hyperglycaemia is a less addressed issue in a paediatric ICU. There are paucity of Indian studies on hyperglycaemia in critically ill children, hence this study is being conducted to find the clinical outcome among those critically ill children, and assess them on basis of PICU stay, inotropes usage, and requirement of mechanical ventilator and on the basis of final outcome, that is survived or did not survive.



OBJECTIVES

OBJECTIVES

PRIMARY OBJECTIVE-

To find out the prevalence of hyperglycaemia in critically ill children admitted in PICU.

SECONDARY OBJECTIVE-

To assess the clinical outcome among hyperglycaemic on the basis of -

- 1) PICU stay
- 2) Inotropes used
- 3) Mechanical ventilation
- 4) Final outcome- discharged healthy or expired.



REVIEW OF LITERATURE

REVIEW OF LITERATURE

Hatred stirs up strife, but love covers all sins”

Proverbs 10: 12

“If hatred is deep within one’s own body as stress compounds like cortisol pour in to the system. These compounds will erode the natural, God given immune system that was created to help us fight off diseases”.

HISTORY

Ebers Papyrus, was an ancient Egyptian text related to the practice of medicine, written back in 1550 BCE. In ancient Greece, the Hippocrates, ‘the father of medicine’, described polyuria and wasting of the body. His follower, Aretaeus of Cappadocia, a Greek physician, was the first person to use the word ‘diabetes’, derived from the Greek word ‘siphon’, in relation to these symptoms.(8,9)

In this parallel time, concepts related to disease, stress homeostasis, were beginning to emerge. Greeks like Heraclitus (540–480BC) and Empedocles (495–435 BC) used words like ‘balance’ and ‘equilibrium’ to define the basics of life. Hippocrates further elaborated by describing health as harmony, and disease as disharmony (10).

In Europe, Thomas Willis (1621–1675), studied medicine at Oxford, defined the word diabetes as the ‘pissing evil’ (11) but also suggested that the condition predominantly was a disease of the blood(8).

Major progress in the concept of stress hyperglycaemia was seen during the 19th century, with the rise of the experimental period in diabetes and work was defined on modern medical views of stress conducted by Claude Bernard (1813–1878) and Walter Cannon (1871–1945). Bernard also described about new and modern ideas on the concept of stress. He also appreciated the fact that, organisms are closely receptive to their external environment, they also aim to maintain a stable and independent internal environment called as ‘Milieu Interieur’. (12)

In 1855, Bernard was the first person to describe about hyperglycaemia in critically ill patients (13).

In the 20th century, after 20 years, Cannon expanded the earlier concept of Bernard’s restating milieu interieur to ‘homeostasis’, (10, 14)

Homeostasis is a maintenance of physiological variables within admissible, narrow ranges, rather than more precise fixed values. (15)

In 21st Century, stress hyperglycaemia has been studied along various common condition admitted in hospitals, conditions like chronic obstructive pulmonary disease (16), pneumonia (17), stroke (18) and sepsis (19). In almost all cases, adverse outcomes have been identified in association with stress hyperglycaemia.

The history of stress hyperglycaemia is very complex and captivating. This knowledge has accumulated over centenary and across various continents, embracing phrases such as homeostasis and ‘fight or flight’. Despite the involvement of a large number of physicians, the spread and acceptance of clinical knowledge has remained negligible. This problem continues till today. Popularising the term ‘stresses within the medical field.

DEFINITION

Stress Hyperglycaemia- The transient increase in plasma glucose levels (usually above 126 mg/dl) during acute illness or physical or psychological stress, which subsides when the stressful condition resolves (20) is termed a stress hyperglycaemia.

Critical illness- Is defined as any severe problem with the airway, breathing or circulation, or acute deterioration of conscious state; including apnoea, upper airway obstruction, hypoxaemia, central cyanosis, severe respiratory distress, total inability to feed, in shock, severe dehydration, actively bleeding requiring transfusion, of the patient is unconscious or having a seizure (21)

Critically Ill Child- A critically ill child is admitted in an ICU on the basis of PAT assessment as per PALS i.e. Paediatric advanced life support.

PAT triangle assesses the initial assessment on the basis of visual and auditory examination. Consisting of Appearance, work of breathing, and circulation (22).



FIG 1- Paediatric Assessment Traingle (PAT)

The Initial Impression Of Pediatrics Assessment Triangle (22)

It's is the first step towards the approach to a sick child. This step begins while the child is being brought into emergency room/opd. It is often referred as "hand off assessment" or doorway assessment. It is a visual or auditory impression of how sick a child is. The complete process is called initial assessment. It usually takes a few seconds.

The three main parameters to decide the urgency to intervene as shown above in the pediatric assessment triangle.

- **APPEARANCE (consciousness)-**

The appearance of the child gives an insight into the overall physiological status and functioning of the brain.

- 1) **TONE-** look at the general posture the child has adopted.
- 2) **INTERACTIVE-** Is the child is responsive and interacting appropriately (normal), unresponsive or dull (lethargic).
- 3) **CONSOLABLE-** crying unusually (irritable), consolable or inconsolable.
- 4) **LOOK/GAZE-** How is the child looking at the mother, is there a vacant gaze (not targeted at any particular object)?
- 5) **SPEECH-** is the child able to speak or vocalise as is appropriate for age or is there a paucity/ weak/ hoarseness of voice.

IDENTIFY- abnormality in any of these parameters points towards a brain dysfunction that is either primary dysfunction or secondary to hypoxia (respiratory or circulatory insufficiency).

- **BREATHING** – in this parameter we look for -

- 1) Whether the child is breathing at all or not ? (apnea)

- 2) Whether the breathing is too fast or too slow ? (bradypnea/tachypnea)
- 3) Are there any audible sounds during respiration ? (stridor,wheeze,gurgling)
- 4) Is there breathing regular and smooth or asynchronous/jerky/paradoxical
- 5) Is there use of any accessory muscles? Flaring of alae nasi, head bobbing ?

IDENTIFY- If abnormalities is present it point towards primary respiratory dysfunction.

- **COLOUR-** it is the third parameter to be observed

- 1) Pale child – indicates anaemia or blood loss.
- 1) Bruises, ecchymosis or petechial spots may indicate bleeding diathesis.
- 2) Mottling or dusky or blue hue over the skin suggests vasomotor instability
- 3) Cyanosis
- 4) Any Evidence of active bleeding.

IDENTIFY- abnormalities in these paramenters are a pointers towards a primary circulatory dysfunction.

PICU CARE FOR CRITICALLY ILL CHILDREN

Two levels of PICU care are identified for sick children, that is level 3 and level 2. Level 3 is called tertiary PICU and can be organized with a level 2 that is step down or high dependency service. In a small private setups, level 3 and level 2 care can be provided in one unit if facilities, equipment or personnel are available.

Admission criteria to level 2 PICU care step down (23) -

Croup (laryngotracheobronchitis) requiring oxygen

Asthma requiring hourly nebulizations/oxygen requirement

All patients requiring more than 50% oxygen to maintain saturations

Closed head/skull fracture

Diabetic ketoacidosis with pH <7.2
Patients with episodes of apnea
Patients with abdominal trauma (renal/hepatic/splenic injury)
Severe dehydration with mental status change
Post-operative patients after major surgery with post-operative pain/blood loss/stress
Patients recovering from critical illness, but requiring close monitoring

Table 1 – Admission Criteria to level 2 PICU care (step down)

Admission criteria for level 3 PICU care (23)-
All patients requiring mechanical ventilation
Patients requiring impending respiratory failure
<ul style="list-style-type: none"> • Upper airway obstruction
<ul style="list-style-type: none"> • Lower airway obstruction
<ul style="list-style-type: none"> • Alveolar disease
<ul style="list-style-type: none"> • Unstable airway
All paediatric patients after successful resuscitation
Comatose patients
<ul style="list-style-type: none"> • Meningitis, encephalitis

• Hepatic encephalopathy
• Cerebral malaria
• Head injury
• Poisonings
• Status epilepticus
All types of shock/haemodynamically unstable patients
• Septic/hypovolemic/cardiogenic/neurogenic shock
• Bleeding emergency such as gastrointestinal bleeding, bleeding diathesis, disseminated intravascular coagulation
• Multiple trauma
Cardiac arrhythmias
• Hypertensive emergencies
• Severe acid base disorders
• Severe electrolyte abnormalities
Acute renal failure
• Patients requiring acute haemodialysis
• Hemofiltration-peritoneal dialysis
Post-operative patients and all post- transplant patients
Patients requiring ECMO
Malignant hyperpyrexia
Acute hepatic failure

Table 2 – Admission Criteria to level 3 PICU care

METABOLIC ABNORMALITIES SEEN IN A SICK CHILD

A sick child manifests plenty of electrolyte abnormalities like changes in serum sodium levels- hyponatremia, hypernatremia. Changes in serum potassium levels- hyperkalaemia, hypokalaemia. Other changes seen are Hypoglycaemia or hyperglycaemia. Hyperglycaemia in grievously ailing paediatric and adult patients, lately has gained considerable amount of attention due to its association with unfavourable clinical outcomes, like elevated frequency

of mortality and morbidity (29)

DISORDERS OF SODIUM

Hyponatremia- defined as serum sodium levels <135 mEq/L, while severe hyponatremia is defined as <120 mEq/L (29)

This may be caused most often by water retention or sodium loss or both. Therefore extracellular fluid volume may be high, normal or low.

Hyponatremia seen in hyperglycaemia is usually Pseudo- hyponatremia. Intravascular compartment is filled with high molecular weight glucose molecules, which causes shift of water from intracellular to extracellular compartment, leading to dilutional hyponatremia.

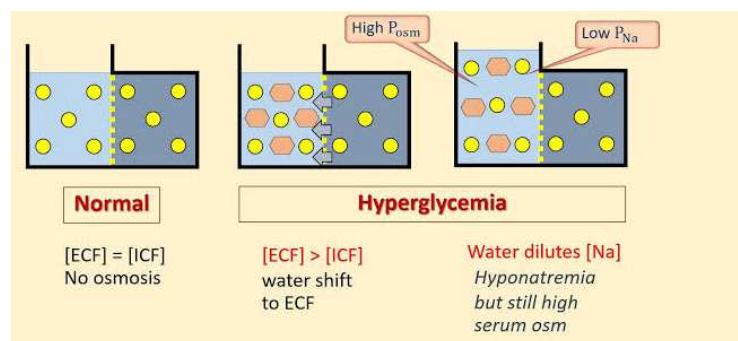


FIG 2- Hyperglycaemia Induced Hyponatremia

Hypernatremia- defined as serum sodium > 145 mEq/L. symptoms usually exceeds when levels are >160 mEq/L.

Hypertonicity either results from excessive loss of body water through kidneys, respiratory tract, skin, gastrointestinal tract and gain body solute, causes life threatening neurological manifestations or due to increase in the osmolarity.

Hypernatremia and hyperglycaemia are two major common causes of hypertonicity. Severe hyperglycaemia developing on the ground of another condition cause hypernatremia and extreme hypertonicity, like seen in case of hyperglycaemic hyperosmolar state, leading to various manifestations like cerebral shrinkage, vascular rupture, decreased contractility of the heart, hyperventilation, cramps as shown in the picture below.

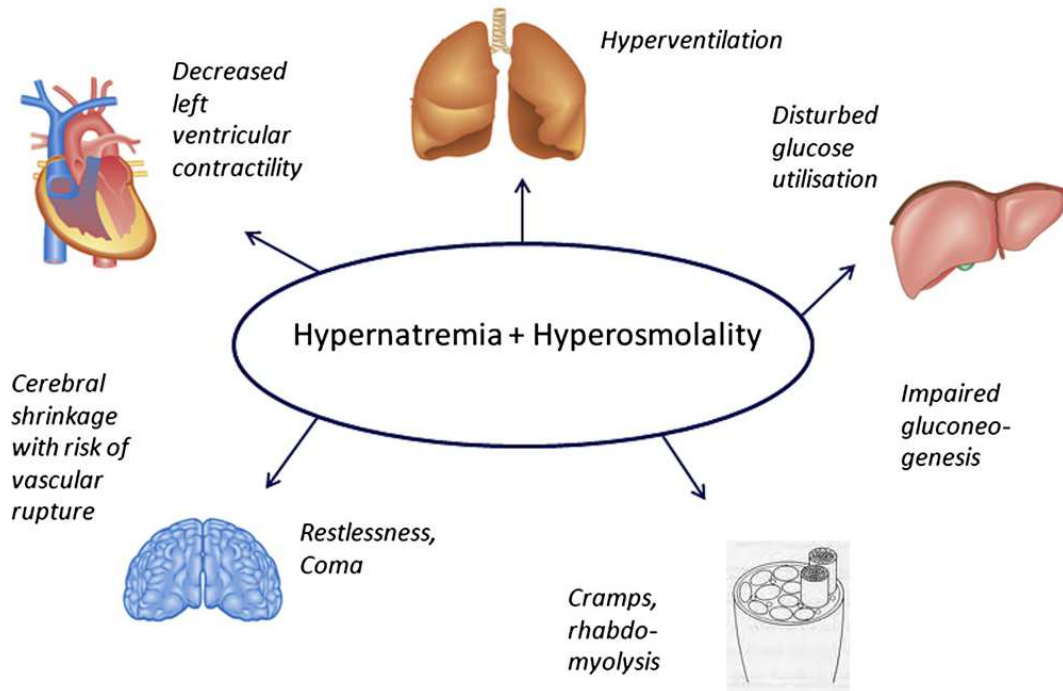


FIG 3- Hypertonicity due to Hyperglycaemia and Hypernatremia.

DISORDERS OF POTTASIIUM

Hyperkalemia- is defined as high serum potassium levels (> 5.5 mEq/L), which further lead to a series of complications. Compared with the general population, patients with hyperglycaemia are more prone to hyperkalemia such as hyporeninemic hypoaldosteronism, hyperosmolality, insulin deficiency (30).

1) Hyporeninemic Hypoaldosteronism-

Hyporeninemic hypoaldosteronism is a syndrome which is caused by a reduction in the synthesis and secretion of renin by juxtaglomerular cells in the kidneys. The RAAS gets dysfunctional due to reduced renin production and synthesis, further leading to a reduction in the secretion of aldosterone by the adrenal glands. Aldosterone is secreted by zona glomerulosa of adrenal gland and acts by promoting the reabsorption of Na^+ and secretion of K^+ into the lumen of the collecting duct, thus aldosterone has a major role in potassium regulation in our body. Hyperglycaemia is the most common risk factor for developing hyporeninemic hypoaldosteronism and there are numerous factors in the hyperglycaemia patient which contributes in the reduction in the release of renin in the body. (30, 31)

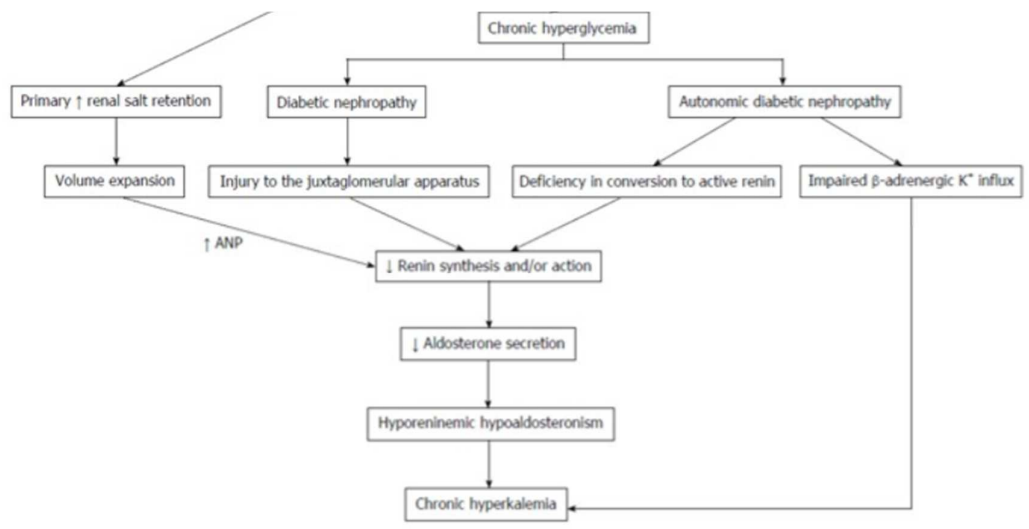


FIG 4- Hyporeninemic Hypoaldosteronism

2) Hyperosmolality

Patients with hyperglycaemia have increased plasma osmolality. Which causes a concentration gradient, the concentration gradient established promotes an outflow of water from inside the cells to the interstitium, and potassium, which is the most abundant

intracellular cation, is carried by the water outside of the cells, elevating its serum concentration (30, 32).

METHODS TO CHECK FOR BLOOD GLUCOSE

The glucometers predominantly work on 2 principles-

- 1) Electrochemical method
- 2) Reflectance photometry

In Electrochemical glucometers, the enzyme used is glucose oxidase which induces an electric current through the strip, which is equally proportionate to the amount of glucose.

In Reflectance glucometers, the strip which is tested, changes colour according to the amount of glucose in the sample. These glucometers quantify the colour change by reflectance photometry.

Laboratory measurements of blood glucose-

There are three basic approaches to the laboratory measurements of blood glucose concentration:

- 1) Reducing methods
- 2) Condensation methods
- 3) enzymatic methods

Reducing methods are the oldest approach of testing blood glucose levels. Reducing properties of glucose change the state of metal ion while glucose gets oxidized. These reducing methods are non-specific, and can react with any strong reducing agent.

The aldehyde group present in the glucose undergo condensation with the aromatic compound and yields a coloured product. This colour is then measured by spectrophotometrically to estimate the glucose concentration. This condensation reaction is

highly corrosive and toxic, hence this method of glucose estimation is not used in clinical laboratories.

In laboratories, most commonly used method is by the enzymatic method. The enzyme glucose oxidase reacts with glucose, water and oxygen and form gluconic acid and hydrogen peroxide. Consumption of oxygen is measured to obtain the amount of glucose present. This glucose oxidase method is relatively inexpensive and specific (1).

Whole vs. Plasma blood glucose- the estimation of whole blood glucose levels are usually 10-15 mg/dl lower than the plasma glucose. Plasma have greater water content than the RBC's or erythrocytes and hence exhibit higher glucose concentration than the whole blood. Another factor which plays a role is hypotension, it results in decreased perfusion and increase in glucose utilization, further resulting in false capillary blood glucose measurements in the hypotensive critically ill children. Several studies showed that finger stick glucose values were lower than the glucose values observed by laboratory glucose measurement methods. Hence on concluding, it can be stated that arterial or venous blood glucose measurements are better than capillary blood glucose (2).



FIG 5.1)- Glucose monitoring by glucometer



FIG 5.2)- Semi-Automated glucose Analyser.

GLUCOSE METABOLISM

Gluconeogenesis-

Gluconeogenesis refers to metabolic reactions, which are regulated both locally and globally (by insulin, glucagon, and cortisol). The purpose of this system, localized in both the cytosol and mitochondria of a cell, is to maintain blood glucose level constant throughout fasting state.

The balance between stimulatory and inhibitory hormones regulates the rate of gluconeogenesis. Liver and secondarily the kidney are the organs that supply circulating blood and consequently, various tissues with glucose. Many tissues depend primarily on glucose to maintain adequate energy levels for their proper function during fasting.

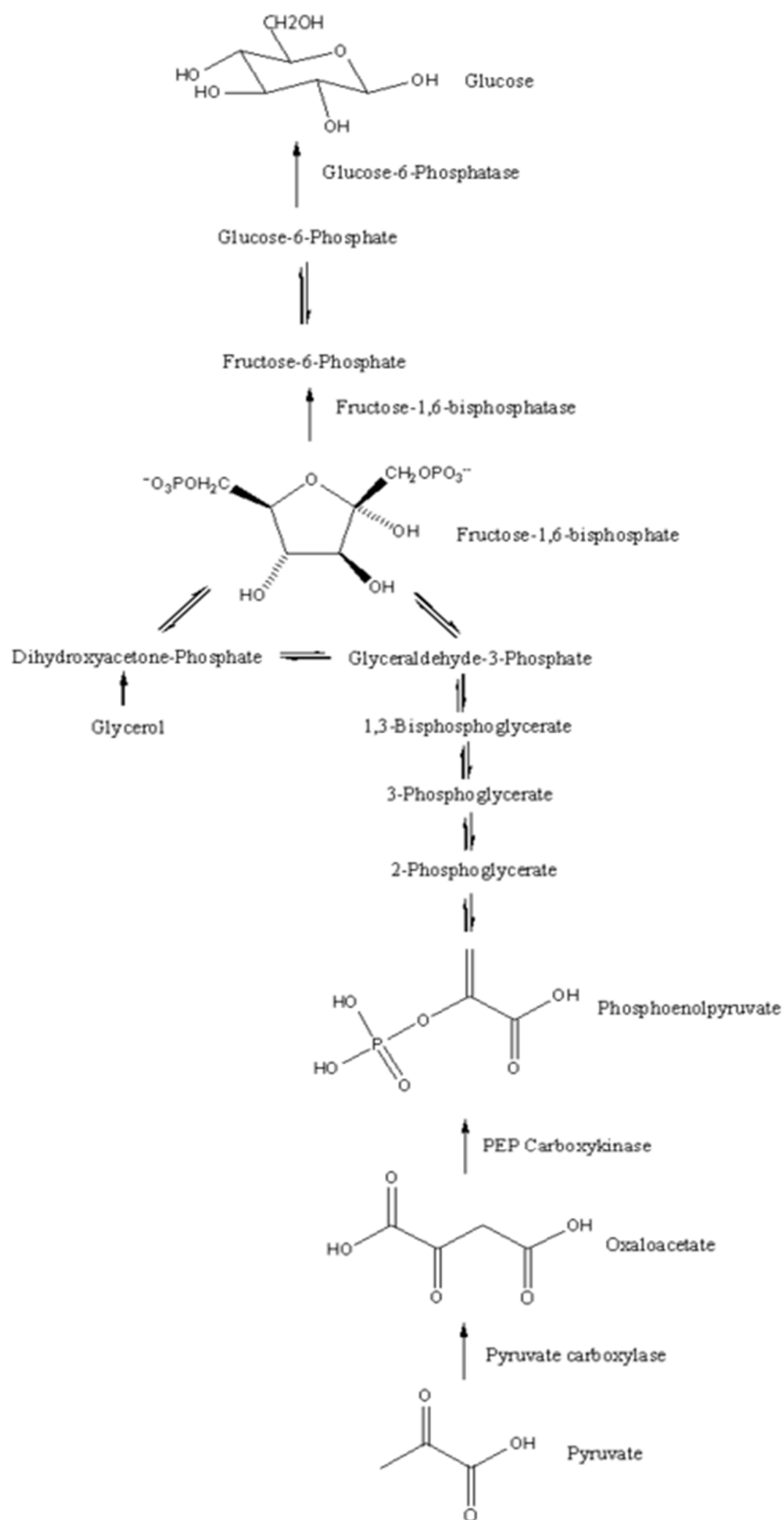


Fig 6- Gluconeogenesis Pathway

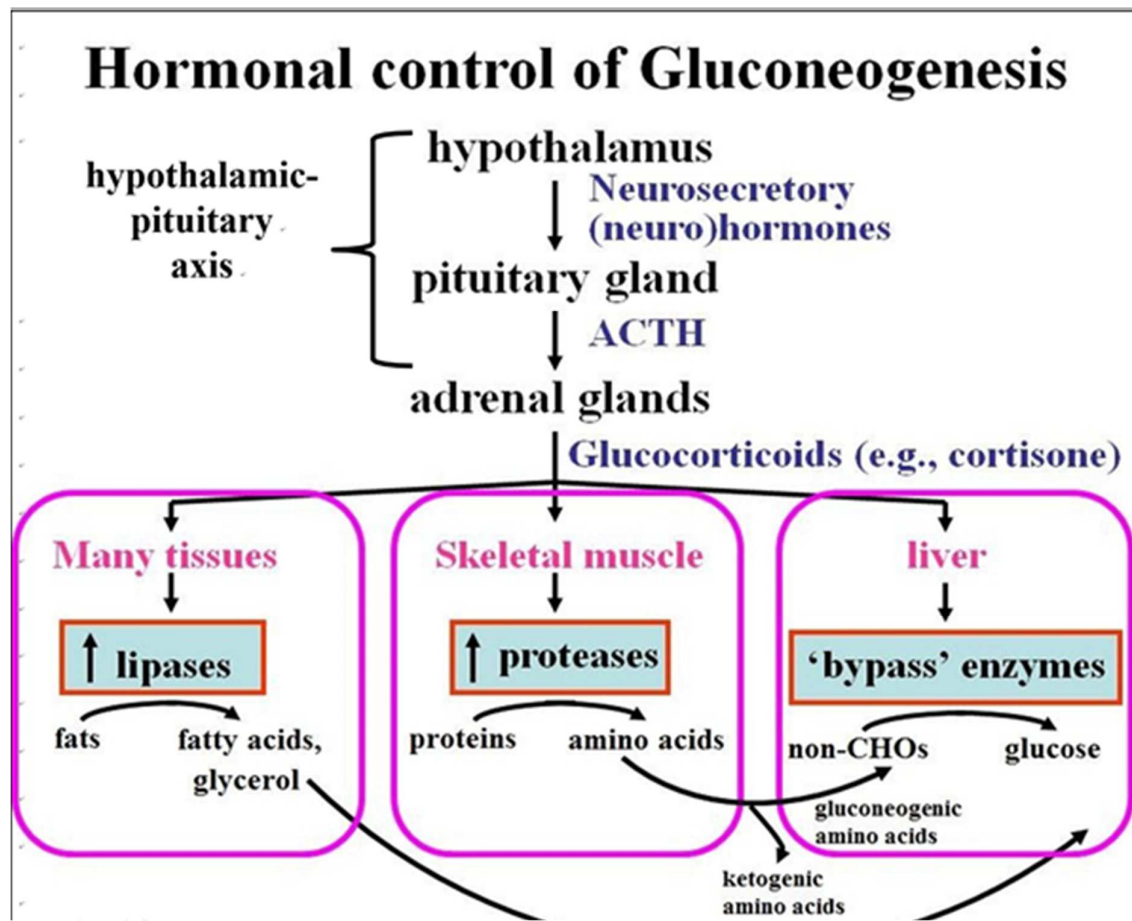


Fig 7- Hormonal control of gluconeogenesis

Stress hyperglycaemia-

Hyperglycaemia has been linked to adverse outcomes in a number of paediatric groups, children experiencing septic shock (30), subjects with traumatic brain lesions (31), beneficiary of skin grafts for severe burn damages (32), and typical PICU subjects (33-35).

Hyperglycaemia also has been linked to unfavourable clinical outcomes in a critically ill child, critical illness is any severe problem with the airway, breathing or circulation, or acute deterioration of conscious state; includes apnoea, upper airway obstruction, hypoxaemia, central cyanosis, severe respiratory distress, total inability to feed, in shock, severe dehydration, actively bleeding requiring transfusion, of the patient is unconscious or having a seizure (34).

It has been previously reported that the length of period of hyperglycaemia was directly and independently associated with heightened rates of mortality and morbidity for paediatric subjects (36). These observations were expanded by the authors by correlating a number of glycaemic ranges with the prevalence of unfavourable sequelae and the development of hypoglycaemia (37).

Insulin therapy aimed at acquiring stringent glycaemic regulation has now turned out to be ubiquitous in several ICUs. Whether the possible benefit of stringent glycaemic regulation is the consequence of avoidance of hyperglycaemia or is linked directly to the influence of insulin dispensation, like enhancement of anabolism and rectification of relative insulin insufficiency, has been the initiator of disagreement (34).

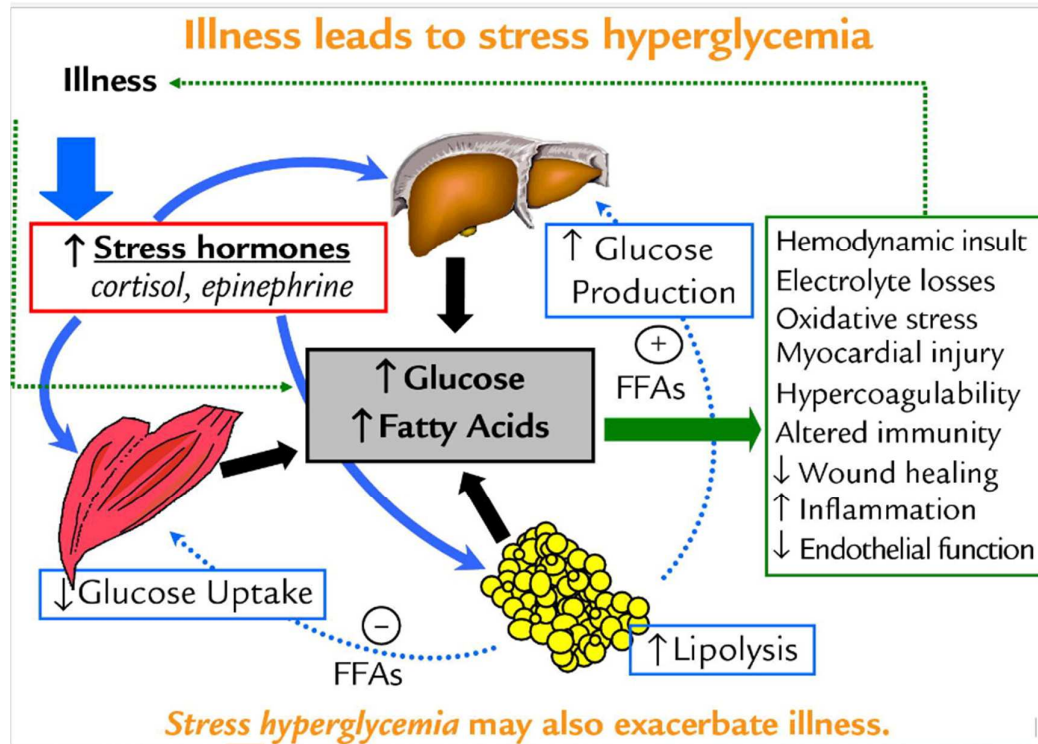


Fig 8 – stress hyperglycaemia

Nevertheless, an enthralling prospective observational investigation aiming to find an association between insulin dispensations with clinical outcomes for grievously ailing children subjects indicated that benefits of mortality were ascribable to glycaemic regulation more than the infused insulin dosage (42).

As stringent glycaemic control approaches consistently begin to pervade into paediatric critical care management strategy, we must without doubt comprehend the risks and advantages of such approaches and perceive that a conclusive investigation documenting the advantage of stringent glycaemic regulation in the paediatric population is still deficient. Due to the normal bio variability of levels of blood glucose over period, the development of unintentional hypoglycaemia in subjects receiving strict glycaemic regulation within a restricted euglycemic target is of critical interest, specifically when we contemplate that its

manifestation might be complicated to identify in critically sick subjects, who frequently are anaesthetized and in a state of neuromuscular hindrance. Hypoglycaemia can have significant consequences, especially in the maturing brain, (41) and has been linked to elevated rates of mortality and morbidity in paediatric subjects (35).

Stress hyperglycaemia generally develop through the course of critical ailments in children, paradoxically in subjects with standard glucose homeostasis at the time of admission (35-37, 39). Previously, stress hyperglycaemia during critical ailment of paediatric subjects was contemplated to be, relatively, an adaptive reaction that enhanced survival or, in the most serious case, insignificant (39, 40). Nonetheless, investigation in children have disputed this statement by indicating that stress hyperglycaemia through the period of critical illness is linked to poor clinical outcomes. (25–30, 36, 40–48). On the basis of the premise that stress hyperglycaemia throughout critical illness is probably detrimental, tight glucose control to normalize the levels of blood glucose has surfaced as a logical but unproven therapeutic strategy to enhance the clinical outcomes in grievously ill children.

Several hypotheses have been proposed to account for the perceived differences in outcomes of these trials. These include inconsistencies in the patient groups, variation in glucose control targets, distinction in obtaining these targets, variances in glucose regulation strategies as well as nutrition delivery, differences in sampling and measurement methods, and difference in the expertise in implementation of the treatment strategy (37).

The community of paediatric critical care workers deal with an even larger conundrum because of the deficit of large-scale clinical research into stringent glucose control in critically sick children. A single-centre investigation of tight glucose control in critically ailing children mainly recuperating from cardiac surgery demonstrated decreases in inflammation and duration of stay in intensive care unit, however at the price of a significant

rise in hyperglycaemia (38). Although most clinicians concur that stress hyperglycaemia is probably detrimental and must be steered clear off in seriously ill children, their major concern is iatrogenic hypoglycaemia and some employ a standardized strategy to tight glucose control (39, 40).

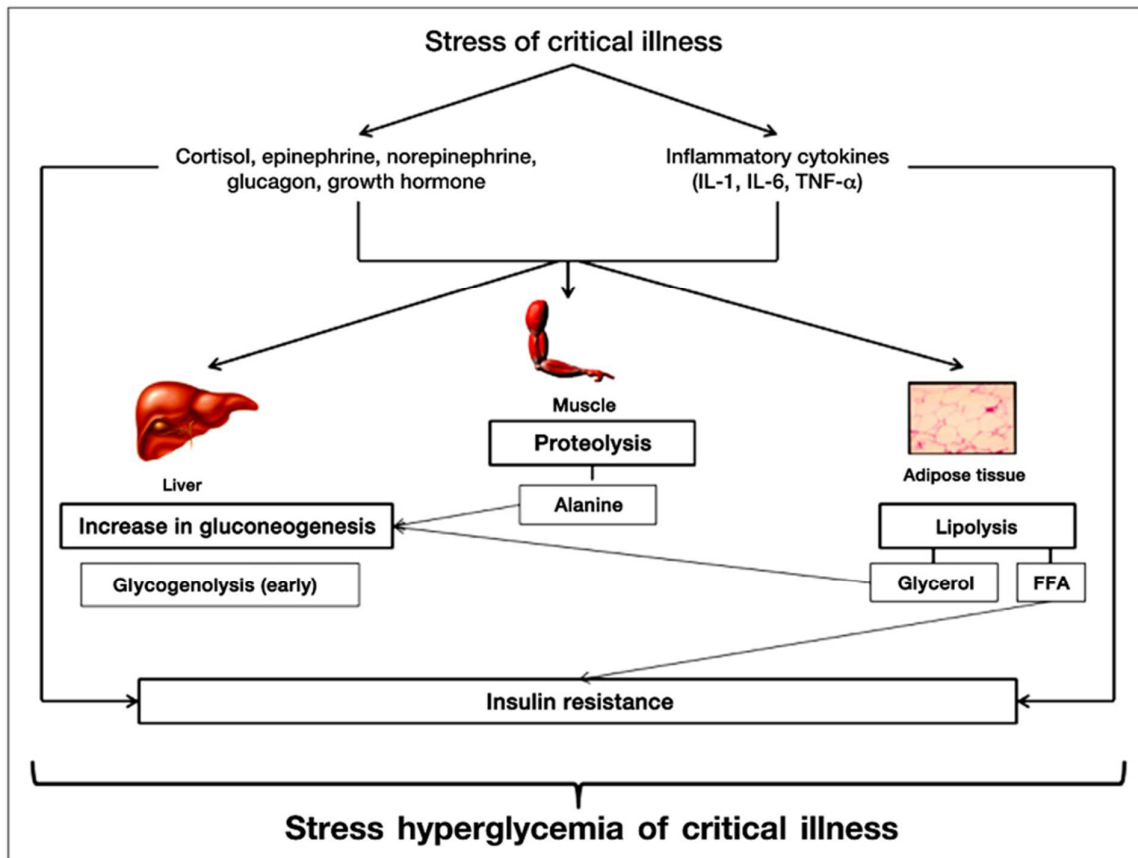


Figure 9: Pathophysiology of stress hyperglycaemia in critical illness. (Adapted from (75))

Pathophysiology of Stress Hyperglycaemia

Grave illness is identified by damage to the cellular milieu from a number of factors like systemic inflammation, oxidative stress, hypoxia, and decreased or redistributed blood circulation. In the backdrop of critical ailment, stress hyperglycaemia evolves mainly via a combination of (1) elevated gluconeogenesis in comparison to glucose clearance and (2) establishment of insulin resistance influencing cellular assimilation of glucose (76) (Figure 6). Both of the aforementioned processes seem to be regulated through elevation of counter modulatory hormones (viz. epinephrine, glucagon, norepinephrine, growth hormone, cortisol) and proinflammatory cytokines [viz. interleukin-1 (IL-1), tumour necrosis factor- α (TNF- α),

interleukin-6 (IL-6)](77,78). Moreover, proinflammatory cytokines can directly hinder insulin production by pancreatic β cells via activation of α adrenergic receptors (79). The general influence of stress hyperglycaemia in critical ailments is to elevate blood glucose concentrations and furnish a prime source of energy for important organs in the body during the period of elevated metabolic demand. While originally stress hyperglycaemia may stand for adaptive feedback by the body through the acute stages of the ailment to enhance the probability of survival, prolonged existence of stress hyperglycaemia throughout chronic ailment can be detrimental.

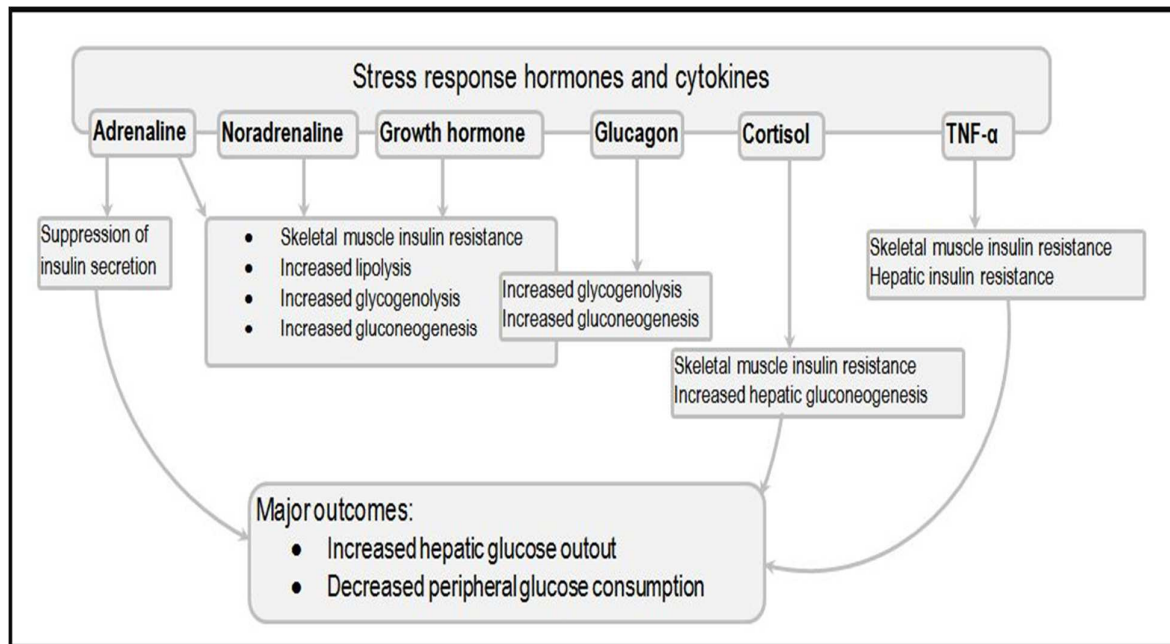


Figure 10- stress response

Stress hyperglycaemia in paediatrics

Few of the pioneering papers pertaining to hyperglycaemia in sick children were case studies from the early 1990s discussing about the increased glucose concentrations in distinct primary diseased conditions like childhood diarrhoea, gastroenteritis or respiratory distress (69–71). ‘Stress hyperglycaemia’ was hypothesised to be linked to the subsequent establishment of conspicuous type 1 diabetes (72, 73). One of the earliest endeavours to establish the frequency of hyperglycaemia in sick children appeared in an investigation conducted in 926 paediatric subjects attending a non-rural emergency division who needed venepuncture for the assessment of their condition; 3.8% of the subjects were observed to maintain a blood glucose concentration of no less than 150 mg/dl. Susceptibility factors for stress hyperglycaemia were high grade fever, which necessitated admission as well as administration of intravenous fluids (74). According to a study from India (30) the frequency of hyperglycaemia (≥ 150 mg/dl) in childhood ailment was reported to be 4.7%, however even though the mortality in the hyperglycaemic category was twice the rate in the normal category, the variation however did not reach statistical significance. It was concluded that hyperglycaemia was frequent, temporary and not clinically consequential. A different investigation (51) inferred that reduced attachment of insulin to receptors and repression of insulin receptors results in the hormonal modifications raising glucose synthesis and peripheral tissue resistance (51).

Several reports pertaining to the investigations dispute the concept that hyperglycaemia is a standard bodily function which is a favourable response towards stress. Grievously ill subjects in the intensive care unit (ICU) environment who are vulnerable to severe and persistent stress frequently experience hyperglycaemia via a number of proposed processes, together with counteracting modulatory hormone-assisted increased activation of the gluconeogenesis and the glycogenolysis pathways, and the decreased activity of glucose transporters accompanied by decreased peripheral usage of glucose by tissues like liver and skeletal muscle (41). Previously, the established advantage of this modified glucose self-regulation was to elevate energy substrate to critical organs like myocardium and the brain, and interfering with the normal increase in glucose quantities was contemplated as damaging (42). Opposition to this perspective have come to light from investigations evaluating mortality and clinical morbidity consequences in addition to investigation concentrating on alterations of both tissue-specific as well as biochemical modifications in animal models as well as patients with varying amounts of glucose and glycaemic regulation (42–46). In paediatric cases, the conventional dread towards hyperglycaemia as an outright hindrance for contemplating stringent glycaemic regulation in the paediatric ICU (PICU) has been investigated.

Mechanisms of cell injury through hyperglycaemia

The hypothesized mechanisms through which hyperglycaemia generate cell injury is detailed in Table (20, 41–53). Even though isolated to assist comprehension, the processes are inter dependent, and their interaction essentially justify how hyperglycaemia presents its influences in vivo as well as how insulin regulated euglycemia can be defensive. The processes mentioned especially in the paediatric reports encompasses oxidative injury and

insufficiency in antioxidant defences in subjects with diabetes type 1 and modified cytokine amounts in paediatric subjects suffering from septic shock and meningococcal sepsis (52,53). Few of the hypothesized advantages of good glycaemic regulation in the ICU are complicated and almost impossible to isolate from the non-dependent influence of insulin. Several clinical reports and experimental studies using animal models established that stringent glycaemic regulation supersede the insulin-controlled advantages (20, 54) and that raise in the dose of insulin was linked to a rise in mortality (21).

Mechanisms for cell injury in the hyperglycaemic state/insulin resistant state
Oxidative stress (damage lipids, proteins, DNA and to the cells via apoptosis)
<ul style="list-style-type: none"> • Increased reactive oxygen species
<ul style="list-style-type: none"> • Polyol pathway
<ul style="list-style-type: none"> • Protein glycation
Decreased antioxidant protection/disposal
<ul style="list-style-type: none"> • Low levels of glutathione peroxidase, superoxide dismutase (enzymatic)
<ul style="list-style-type: none"> • Low levels of plasma antioxidant capacity, vitamins C,E,A (nonenzymatic)
Increased rate of muscle protein catabolism
<ul style="list-style-type: none"> • Increased phenylalanine release
Altered balance in immune system regulation
<ul style="list-style-type: none"> • Decreased innate immunity
1) impaired phagocytosis
2) impaired neutrophil function
3) increased deactivation of monocytes and neutrophils during infection
<ul style="list-style-type: none"> • excessive and unchecked cytokine production and increased C- reactive protein
<ul style="list-style-type: none"> • IL-6, TNF- alpha, IL-8
Association with dyslipidemia of the critically ill
<ul style="list-style-type: none"> • Elevated triglycerides, elevated VLDL, decreased LDL and HDL
Cardiac dysfunction
<ul style="list-style-type: none"> • Nitric oxide mediated monocyte damage

<ul style="list-style-type: none"> • Reactive oxygen species mediated myocardial damage
<ul style="list-style-type: none"> • Increased angiotensin-II
<ul style="list-style-type: none"> • Increased systemic vascular resistance
<ul style="list-style-type: none"> • Decreased cardiac output, cardiac index, stroke volume
Cerebral ischemia
<ul style="list-style-type: none"> • Intercellular acidosis, increased brain edema, disruption of blood-brain barrier
Endothelial activation and damage
<ul style="list-style-type: none"> • Excessive nitric oxide concentration can be proinflammatory and cause ischemia and cell damage.
<ul style="list-style-type: none"> • Activated leukocytes release reactive oxygen species
<ul style="list-style-type: none"> • Excessive leukocyte adhesion hamper perfusion
Mitochondrial abnormalities
<ul style="list-style-type: none"> • Dysfunction in respiratory chain and energy production
<ul style="list-style-type: none"> • Ultrastructural damage to hepatocyte mitochondria

Table 3: Compilation of mechanisms for cell injury in the hyperglycaemic/insulin resistant state (Adapted from (55))

Hyperglycaemia in the intensive care unit

Retrospective investigations evaluating grievously ailing subjects revealed that hyperglycaemia was widespread, as well as that hyperglycaemia during the admission time or shortly after the process was linked to poor clinical consequences (56–58). Prospective investigations inferred that hyperglycaemia gave rise to more severe outcome with respect to the duration of stay, rates of infection, mortality and additional morbidities (2,59,60). Exhaustive insulin regulation enhanced clinical sequelae, inclusive of mortality, in grievously sick surgical subjects (3), however, the influence of glycaemic regulation in medical ICU with respect to reduced mortality was not established(4). An overview of several investigations pertaining to the subject was featured in the American College of

Endocrinology and American Diabetes Association Consensus Conference in 2006. A deliberation of the affirmatory reports that applied glucose regulation in combined surgical/medical ICU settings (61).

Challenges in paediatrics

In grievously ill paediatric cases, the disordered physiological feedback to distress and the consequences of hyperglycaemia on tissues are not extensively characterized. Moreover, majority of the research designs associating hyperglycaemia to end-result measures fall short of demonstrating causality. It likely possible to employ processes of hyperglycaemia associated tissue injury evidenced in the reports pertaining to adult subjects to paediatric cases, however, investigations are in the initial stages of testing this argument. It may also be admissible to employ adult objective ranges for glycaemic regulation to paediatric subjects. Nevertheless, PICU subjects, in entirety, are in better health than the adult subjects, in general the mortality rates at PICU are remarkably lesser than adult ICU mortality rates (62–64), and on an average a child's tissue and other supportive structures (encompassing vasculature, blood cells, kidney, liver and muscle) have not yet been affected by the adult levels of lipotoxic, oxidative and hyperglycaemic damage by the absolute essence of duration of exposure in the case of adult subjects (65).

Moreover, a dilemma exists with respect to the admissible age-linked standards for euglycemia in sick children giving rise to hesitation to implement stringent glucose regulation, and the possibility of complications associated with hyperglycaemia as well as hypoglycaemia is greatest in the youngest paediatric cases below 3–5 years who are going through crucial brain development. In adult subjects, when a stringent glucose regulation

approach implemented in the surgical ICU was employed in the medical ICU, occurrence of hypoglycaemia was more frequent and was a self-standing risk component for mortality(4).

Modifications in Glucose Metabolism

In the course of being healthy, a stabilised synergy of glycogenolysis and gluconeogenesis conserve adequate blood glucose levels between meals. Subsequent to a meal, increased blood glucose concentrations lead to insulin production along with repression of gluconeogenesis and elevated synthesis of glycogen. Through the state of stress, elevated levels of counter modulatory hormones and proinflammatory cytokines primarily regulate rapid glycogenolysis and gluconeogenesis, leading to increased levels of blood glucose (76). Glycogen reserves are swiftly exhausted in the unfed condition, with glycogenolysis chipping in to restricted glucose synthesis (78). Nevertheless, hepatic gluconeogenesis continue to exist, leading to elevated glucose synthesis and occurrence of stress hyperglycaemia (80,81). The efflux of catecholamines throughout critical ailment leads to elevated concentrations of glucagon so as to maintain gluconeogenesis even while there is presence of increased insulin levels (82). The kidney too is a critical reservoir of gluconeogenesis in severe illness and can account for almost 40% of glucose synthesis as feedback to catecholamines (83). Alternate hormonal modifications, like rise in growth hormone and decrease in insulin-like growth factor-1, assist the disintegration of muscle to liberate alanine to the liver to aid in the maintenance of gluconeogenesis (84).

Modifications in Insulin Sensitivity and Secretion

The characteristic feature of a critical illness is the occurrence of both peripheral and central insulin resistance. Central insulin resistance in liver is regulated by epinephrine, glucagon, and cortisol, leading to perpetual hepatic gluconeogenesis despite the presence of increased

concentrations of insulin (82). Additionally hepatic insulin resistance linked to rise in growth hormone and decrease in levels of insulin-like growth factor-1(84). Peripheral insulin resistance develops in adipose tissue and muscle because of modifications in the insulin feedback pathway regulated hugely by counter modulatory hormones and inflammatory cytokines. Elevated levels of growth hormone, cortisol, and epinephrine in grievous ailments hinder the movement of the insulin-dependent glucose transporter protein 4 (GLUT-4) from the internal membrane reserves and decrease insulin binding (85, 86). Inflammatory cytokines influence serine phosphorylation of insulin receptor substrate 1 and impede insulin receptor tyrosine kinase, and as a result decreasing cellular assimilation of glucose through GLUT-4 (87, 88). Elevated levels of free fatty acid (FFA) as a result of lipolysis regulated by growth hormone and catecholamines additionally escalate insulin resistance (87, 88). The peripheral insulin resistance attained in such manner can persist for a prolonged duration subsequent to recovery from grievous illness in paediatric subjects (89). Besides the development of insulin resistance, investigations have also evidenced anomalies in pancreatic β -cell function and decreased insulin release in grievously ailing children (52, 90).

Hyperglycaemia in critically ill children

Among the limited reports on the topic, researchers indicate that hyperglycaemia prevail often in grievously sick children (7–10, 12, 13, 91). Numerous investigations are centred on subgroups of PICU subjects with particular injury or pathological condition. According to two retrospective investigations of paediatric head injury subjects, increased glucose was linked to unfavourable outcomes, with continued hyperglycaemia (described as hyperglycaemia past 24 hours of PICU admission), fulfilling the role as an critical negative predictive factor (9,91). Both investigations employed the Glasgow Coma Scale and the

Glasgow Outcome Scale to categorise subjects by intensity of injury and intensity of the sequelae, separately. In the previous investigation evaluating 50 subjects, glucose was greatly associated with injury intensity (131.5 ± 36 mg/dl in the mild class compared to 237.8 ± 92 mg/dl in the severe class), and there existed a negative association between initial concentration of glucose and clinical after effects (169.79 ± 75.78 mg/dl in the better after effects class compared to 280 ± 77.9 mg/dl in the worse sequelae class). In the severe damage category, a higher mean initial glucose concentration was exhibited in the worse sequelae class in comparison to the better sequelae class. Nevertheless, in subjects with continuously increased glucose concentrations, two-thirds exhibited low scores of outcomes. Utilisation of infusions containing glucose, corticosteroids and anticonvulsants were explored, and 'no interference with blood glucose values' was observed (91). In a 2003 report comprising of 170 children, it was observed that out of 16 subjects who succumbed to death, seven had glucose levels of not less 300 mg/dl at the time of admission, stipulating a threshold past which the level of glucose is majorly linked to death. During the subclass analysis in the aforementioned study, among the most grievously injured, an association in a similar manner between level of glucose at the time of hospital admission and sequelae was observed (9). A 2004 retrospective investigation looking into the levels of blood glucose in 88 NICU cases admitted due to necrotizing enterocolitis exhibited a link between a maximum level of glucose of above 215 mg/dl (11.9 mmol/l) and delayed mortality and duration of stay in ICU (7). Yet another retrospective investigation in 2004 was conducted in 152 severely ill paediatric subjects who were administered vasoactive infusions or mechanical ventilation. Hyperglycaemia was described as above 126 mg/dl in the study. Variations associated with the timing and range of hyperglycaemia, instead of the absolute value of glucose level, were linked to the outcome. Especially, non-survivors exhibited an elevated mean level of glucose at 24 hour compared to survivors, while glucose level above 150 mg/dl at 24 hour resulted in

nearly 3.5-fold rise in risk of mortality. Additionally, period of hyperglycaemia was prolonged in non-survivors when compared to survivors ($71 \pm 14\%$ of days and $37 \pm 5\%$ respectively, $P < 0.001$), and on the condition of being exposed to hyperglycaemia for further than half of the subject's total stay in PICU, the risk of mortality increased to almost six fold in comparison to being exposed to lower than half of the total duration spend in PICU. However, in subjects who were administered insulin, no statistically significant observation was made with respect to the risk of developing hypoglycaemia (12). Nevertheless medications, infusions and nutrition were not accounted for in the study.

Investigative reports on critically ill children

The cases of septic shock in paediatric subjects have encountered contemporary progress in therapy approaches. A longitudinal study comprising of 57 subjects with septic shock resistant to volume, the peak value of glucose levels was estimated and grievousness of illness was assessed using Paediatric Risk of Mortality (PRISM) II score. Details regarding caloric intake, medications, inclusive of corticosteroids, and infusions, were catalogued. The highest glucose level was remarkably elevated in non-survivors compared to survivors (262 ± 110 mg/dl and 167.8 ± 55 mg/ dl respectively, $P < 0.01$), and the highest level of glucose of above 178 mg/dl was linked to 2.59 times raise in risk of demise (confidence interval 1.37–4.88) (8). According to a retrospective investigation associating preliminary, peak and period of hyperglycaemia to sequelae in postoperative paediatric cardiac cases, hyperglycaemia was linked to longer duration of stay ($P < 0.001$), prolonged ventilator use ($P < 0.001$) and loss of life ($P < 0.002$), amidst other variable parameters. The specifics of the medications, inclusive of insulin, corticosteroids, and inotropes, was documented and evaluated. It was accepted,

nevertheless, that the distinction in consumption of the medication between non-survivors and survivors may account for glucose variations, in addition to the event that glucose dispensation through parenteral and enteral methods was not evaluated (19). A retrospective investigation (92) conducted in 37 preterm very low birth weight children who were administered total parenteral nutrition (TPN) observed elevated average maximum levels of glucose in non-surviving infants (241 ± 46 mg/dl versus 141 ± 47 mg/dl, $P < 0.0001$) and longer duration of stay in hospital in surviving infants with elevated glucose levels ($P = 0.006$). An alternate investigation (13) retrospectively evaluated glucose variation in 1094 PICU subjects, inferring that hyperglycaemia, hypoglycaemia and glucose variation were linked to prolonged duration of hospital stay and mortality. An observational longitudinal investigation evaluating children suffering from meningococcal sepsis side by side with children suffering from meningococcal septic shock (MSS) effectively examined levels of glucose, numerous glucoregulatory hormones (insulin-like growth factor-1, insulin, cortisol, glucagons, growth hormone, leptin,) and several assessments of cytokine levels. If the children's pathologic state of hormonal imbalance in critical ailment will mirror the case in adults was the question of interest here, and additionally if distinct phases of this quickly evolving septic impression in children result in distinct hormonal regulation. No differences exist between categories with respect to the glucose level at the time of admission, however ensuing and maximum levels of glucose were more in MSS subjects than in meningococcal sepsis subjects ($P < 0.05$) and associated with the intensity of the ailment ($r = 0.833$, $P < 0.001$). It is interesting to know that, levels of insulin in MSS subjects were remarkably lesser (7.2 versus 19.0 mU/l, $P < 0.001$), indicating hyperinsulinemia in comparison to the insulin resistance observed in the subjects with meningococcal sepsis, and levels of insulin were inversely associated to the amount of soluble cytokine receptors, proposing that the inflammatory flare up plays a role in insulin repression (52). An alternate retrospective

investigation comprising of 942 subjects using several cut-offs for hyperglycaemia to assist in comparing (120, 150 and 200 mg/dl) of the maximum level of glucose within 24 hours and within 10 days was associated with in-hospital mortality and prolonged duration of stay, however, preliminary level of glucose was not associated with risk of demise. An association was observed between the risk of demise and glucose level of over 150 mg/dl (11).

Hyperglycaemia develop often in ICUs and has been dynamically linked to a rise in mortality and morbidity frequency in adults (2, 3, and 5) as well as children (12, 13, and 25). Stringent glycaemic control with insulin dispensation was revealed to decrease the frequency of morbidity and mortality remarkably for adult subjects enrolled in surgical ICU (3). Similar strategy exhibited reduction in frequency of morbidity but not that of mortality in subjects enrolled in medical ICU (4). Falco et al., previously observed that the extent of hyperglycaemia in children subsequent to surgical restoration or palliation of inborn heart anomalies was linked firmly and independently with elevated frequency of morbidity and mortality (14). They also documented that the frequency of mortality was increased among subjects with severe hyperglycaemia, in comparison to those with mild or moderate hyperglycaemia (14).

Even though strict glycaemic regulation has been linked to improved clinical results in the adult subjects, its effect in paediatric subjects has not been investigated extensively. However, there is a high probability that the approach applied for glycaemic regulation in grievously ill adult subjects might not be suitable for the width of the range of age of paediatric subjects. There exists apprehension that glycaemic regulation directed at averting hyperglycaemia meanwhile supporting a stringent euglycemic target, might place subjects at elevated risk for hypoglycaemia. As a matter of fact, a pilot clinical study focussing on intensive insulin treatment administered to adult subjects with extreme sepsis had to be

terminated prematurely due to the increased incidence of severe hypoglycaemia in subjects recruited into the intensive insulin therapy category (22).

As a number of institutions initiated the evaluation of glycaemic regulation approaches for application in the PICU, apprehension in regard to the development of unpremeditated hypoglycaemia and how to avert it should be paramount in the formulation of any investigative protocol, especially while considering the possibly extreme effects of hypoglycaemia on the growing and maturing brain of infants and neonates (23, 24). It has been hypothesized in some studies that a more non-restrictive glycaemic quarry would be linked to a reduced occurrence of hypoglycaemia but not with an elevated frequency of mortality for grievously ill paediatric subjects after surgical restoration or palliation of inborn heart abnormalities.



METHODOLOGY

METHODOLOGY

This study was conducted from January 2021 to December 2021 at KLES Dr Prabhakar Kore Hospital and Medical Research Centre, Belagavi, Karnataka.

Study Design-

Cross-sectional study

Study Duration-

One year duration from January 2021 to December 2021.

Place of Study-

Children between 1 month to 18 years admitted in PICU of KLE'S Dr. Prabhakar Kore Hospital and Medical Research Centre, Belagavi.

Inclusion Criteria-

1) Children between 1 month-18 years who are critically ill admitted in PICU are included in study

Exclusion criteria-

- 1) Children with pre existing or newly diagnosed case of diabetes mellitus.
- 2) PICU stay for less than 24 hours.

3) Children who received drugs like quinine, chloroquine, cyclosporine from outside hospital

Formula used for sample size calculation is

$n = \frac{p(100 - p)Z^2}{E^2}$ n is the sample size required

p is the percentage occurrence of a state or condition (proportion or prevalence),

E is the percentage maximum error required,

Z is the value corresponding to level of confidence required.

Prevalence of euglycemia reported as 24.5%. This prevalence is used for sample size calculation and percentage of maximum error required is 5% at 95% confidence level sample size is given by,

$$n = \frac{24.5 \times (100 - 24.5) \times (1.96)^2}{25}$$

Hence, minimum sample size required is 284.

Methodology-

Children who meet the inclusion and exclusion criteria will be enrolled into the study after taking written informed consent from parents.

A detailed history, demographic data, thorough physical examination will be performed.

Patient will be followed throughout the duration of PICU stay and outcome.

The details of patient on mechanical ventilation, vasopressor use, infusions, steroids and other medications will be recorded.

In all cases blood glucose estimation will be done in laboratory by hexokinase method.

First at admission then every 121 hourly in all cases. If the child falls under hyperglycaemic value (126 mg/dl) the child will be monitored every 6th hourly till the glucose level falls <126mg/dl.

Hyperglycaemia will be considered with blood glucose 126mg/dl.

Hypoglycaemia is 60mg/dl blood glucose,

Euglycaemia is blood glucose between 61 to 126mg/dl as per WHOC 81.

Other lab investigations that are ABG, PT, Aptt, total bilirubin, potassium, CBC, n calcium, which are required for PRISM III score will be performed.

Prism III score will be measured initially after admission and after 4 days in PICU to predict mortality and severity and correlate with blood glucose levels

PATIENT DATA:

All the patients enrolled in the study were subjected to following-

Focused history, past medical history, last meal history, co-morbidities, lab investigations- arterial blood gas, serum electrolytes, total bilirubin, glucose measurement by hexokinase method.

DATA ANALYSIS:

Continuous variables will be given in mean \pm SD/median (range). Categorical variables will be represented by frequency. To check the dependency between attributes Chi- square test will be used. To compare mean/distribution over groups test/ANOVA/Mann-Whitney test/Kruskal-

Wallis test will be used. Logistic regression model will be used to check the potential factors which affects outcome. To check the normality of variables Quantile-Quantile (QQ) plot/Shapiro-Wilk's test will be used. P-value less than or equal to 0.05 shows statistical significance



RESULTS

RESULT

The data contains measurement on 284 subjects whose age ranges from 1 month to 18 years with mean age of 5.53 ± 5.14 years. The following table gives the distribution of subjects according to demographic characteristics.

Table 4: Distribution of subjects according to age. Out of 284 patients, 30.28% are in age from 1 month to 12 months, 46.13% are in 1 to 10 years age group and 23.59% are above 10 years of age.

Variables	Sub Category	Number of subjects (%)
Age	1month-12 months	86 (30.28%)
	1year - 10 years	131 (46.13%)
	> 10 years	67 (23.59%)
Age (years)	Mean \pm SD	5.53 ± 5.14
	Median (Min, Max)	4 (0.08, 17)

Variable	Sub Category	Number of subjects (%)
Gender	Female	119 (41.9%)
	Male	165 (58.1%)
Weight (Kg)	Mean \pm SD	18.36 ± 15.9
	Median (Min, Max)	12 (1.85, 75)

Table 5- distribution of subjects according to gender and weight.

Out of 284 subjects, 58.1% were males and 41.9% were females with gender ratio of 1.39:1.

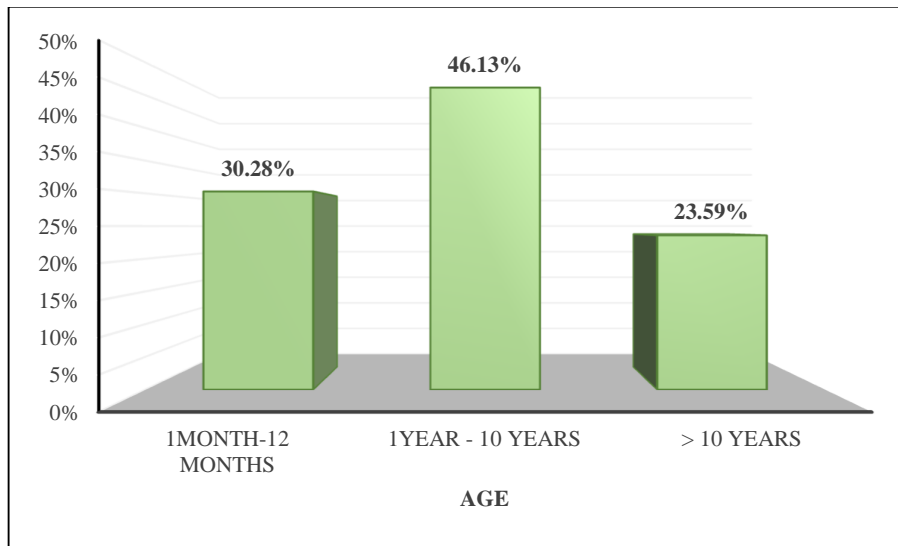


Figure 11: Distribution of subjects according to age.

We got 86 patients (30.2%) in the age of 1 to 12 months of age, 131 (46.13%) patients between 1 to 10 years and patients above 10 years of age were 67 (23.59%).

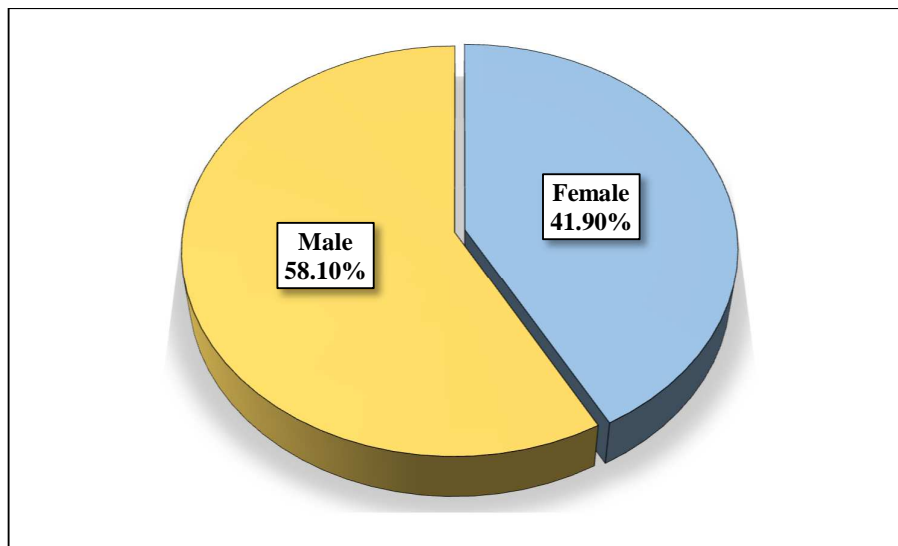


Figure 12: Distribution of subjects according to gender.

In this study out of 284 patients, 58.10% were male and 41.90% patients were female.

The following table gives the distribution of subjects according to glycaemic level.

Table 6: Distribution of subjects according to glycaemic level.

Glycaemic level	Number of subjects (%)
Euglycaemia	163 (57.39%)
Hypoglycaemia	2 (0.7%)
Hyperglycaemia	119 (41.9%)
Mean \pm SD	145.46 \pm 67.25
Median (Min, Max)	121 (11, 414)

Out of 284 subjects, 163 patients (57.39%) have Euglycaemia, 119 patients (41.9%) have hyperglycaemia and 2 patients (0.7%) have hypoglycaemia.

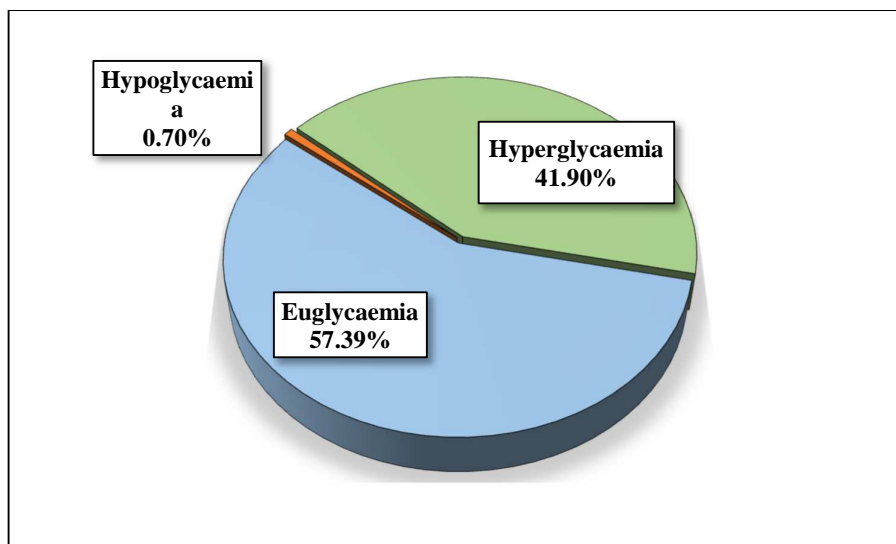


Figure 13: Distribution of subjects according to glycaemia levels.

The following table gives the distribution of subjects with hyperglycaemia levels.

Table 7: Distribution of subjects with hyperglycaemia levels.

Hyperglycaemic level	Number of subjects (%)
126-150mg/dl	38 (31.93%)
150-200mg/dl	30 (25.21%)
>200mg/dl	51 (42.86%)

Out of 119 subjects with hyperglycaemia, 42.86% have > 200mg/dl, 31.93% have >126mg/dl and 25.21% have >150mg/dl.

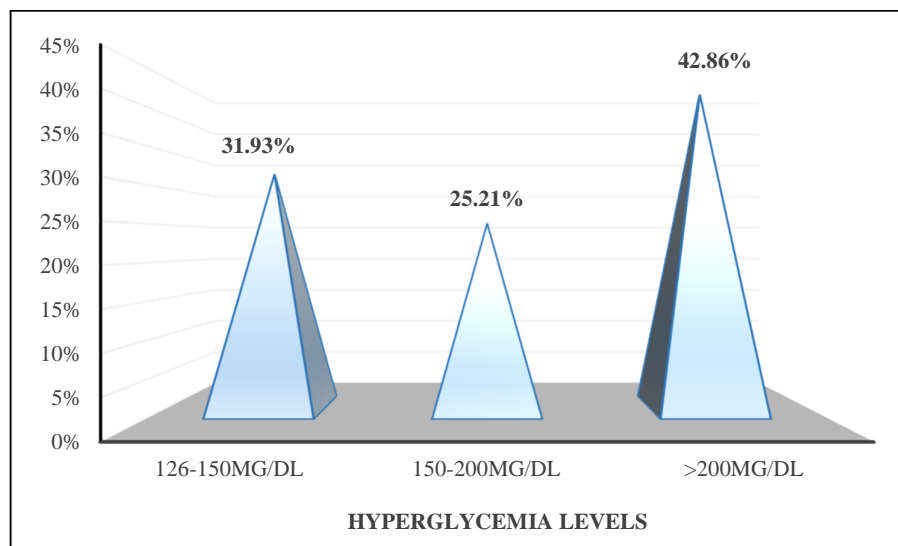


Figure 14: Distribution of subjects according to hyperglycaemia levels.

The following table gives the association of glycaemia levels with age.

Table 8: Association of glycaemia levels with age.

Age	Glycaemia levels			p-value
	Euglycaemia	Hypoglycaemia	Hyperglycaemia	
1month-12 months	41 (47.67%)	0	45 (52.33%)	0.0125^{MC*}
1year - 10 years	87 (66.41%)	2 (1.53%)	42 (32.06%)	
> 10 years	35 (52.24%)	0	32 (47.76%)	

Abbreviation: MC – Chi square test with Monte Carlo simulation, * indicates statistical significance.

From Chi square test, it is observed that, there is significant association of glycaemia levels with age.

The prevalence of hyperglycaemia among the subjects in age group 1month-12 months is 52.33%, in age group 1year - 10 years is 32.06% and in age group > 10 years is 47.76%.

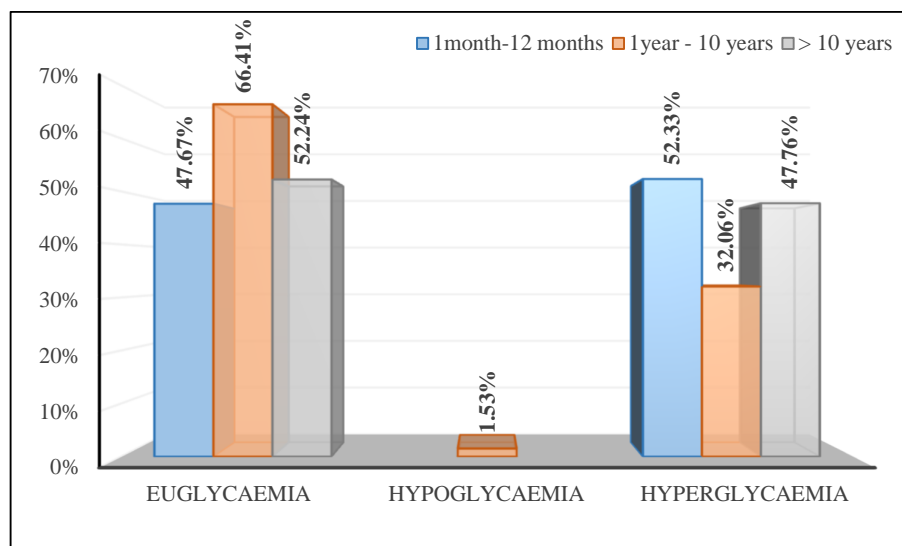


Figure 15: Distribution of subjects according to hyperglycaemia levels and age.

The following table gives the distribution of length of PICU stay.

Table 9: Distribution of length of PICU stay.

Variable	Mean \pm SD	Median (Min, Max)
Length of PICU stay	6.31 \pm 6.07	5 (0.29, 74)

The length of PICU stay ranges from 7 hours to 74 days with mean length of PICU stay of 6.31 \pm 6.07 days.

The following table gives the distribution of system involved.

Table 10: Distribution of system involved.

System involved	Number of subjects (%)
Sepsis	88 (30.9%)
Respiratory	58 (20.422%)
CNS	51 (17.9%)
Cardiology	44 (15.4%)
GIT	22 (7.7%)
Haematology	14 (4.9%)
Trauma	7 (2.46%)

Out of 284 subjects, 30.9% sepsis, 20.42% respiratory, 17.9% were CNS and 15.5% cardiology, 7.7% GIT, 4.9% cases of haematology and 2.6 cases of trauma.

The following table gives the distribution of subjects according to mechanical ventilator

Table 11: Distribution of subjects according to mechanical ventilator.

Variables	Sub Category	Number of subjects (%)
Mechanical Ventilator	No	205 (72.18%)
	Yes	79 (27.82%)

Out of 284 subjects, 79 patients (27.82%) required mechanical ventilator and 94 patients (33.1%) required vasopressors.

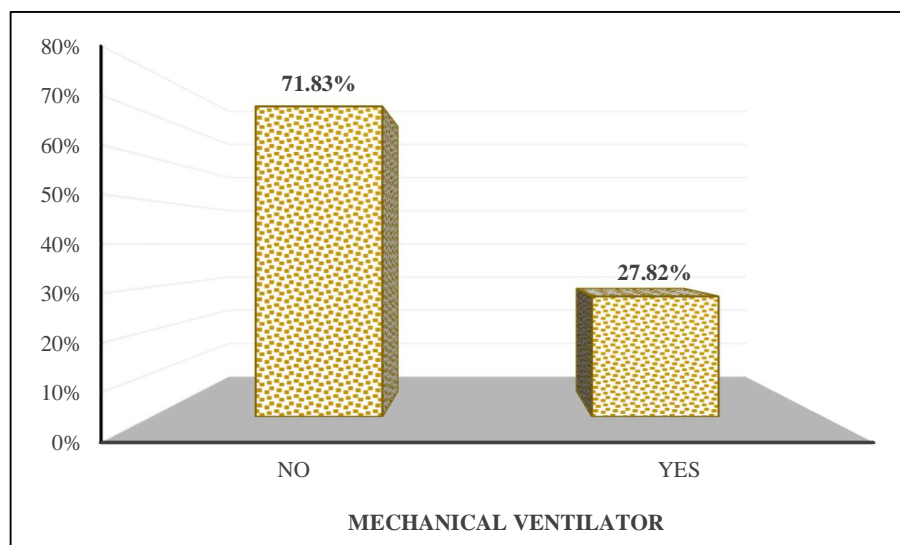


Figure 16: Distribution of subjects according to mechanical ventilator.

Table 12: Distribution of subjects according to inotropes.

Variables	Sub Category	No. of subjects %
Inotropes	No	190 (66.9%)
	Yes	94 (33.1%)

Out of 284 subjects, 94 patients (33.1%) required vasopressors.

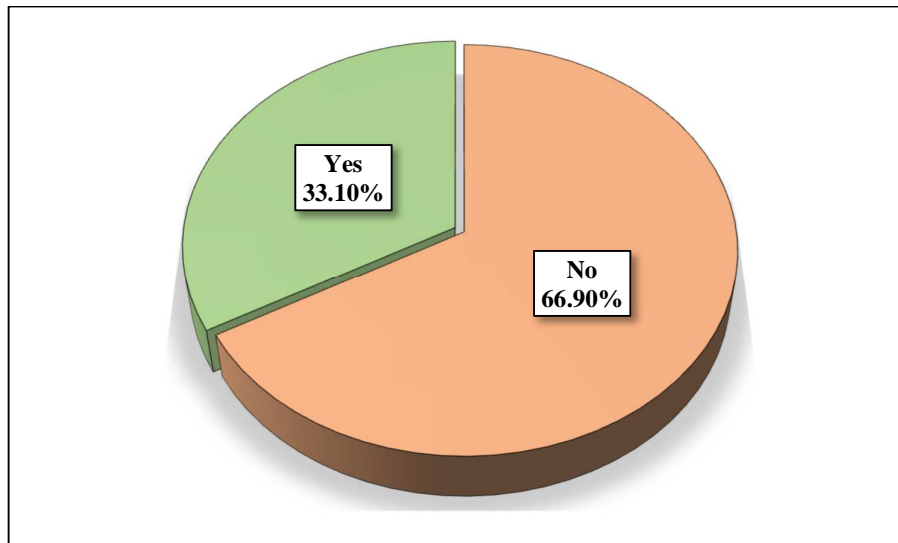


Figure 17: Distribution of subjects according to inotropes.

The following table gives the distribution of subjects according to outcome.

Table 13: Distribution of subjects according to outcome.

Outcome	Number of subjects (%)
Non-survived	72 (25.35%)
Survived	212 (74.65%)

Out of 284 subjects, 74.65% survived whereas 25.35% did not survive.

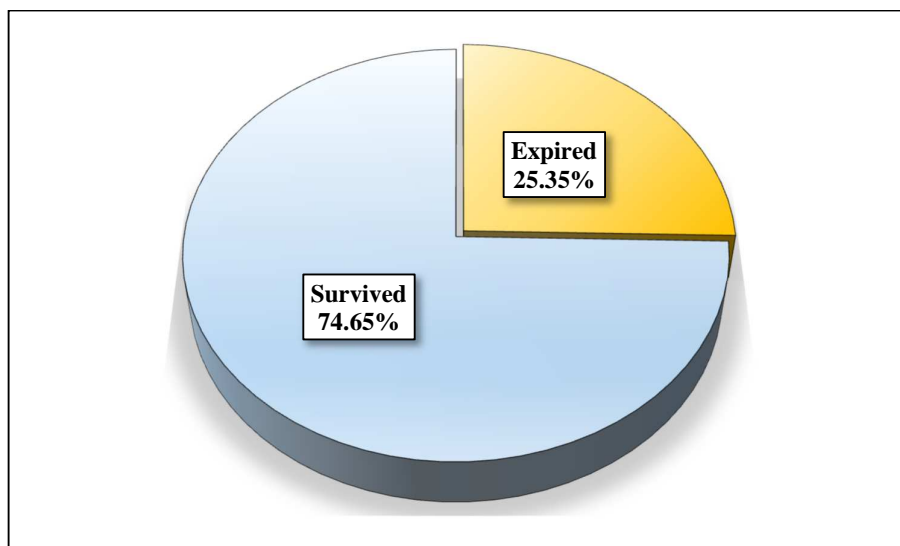


Figure 18: Distribution of subjects according to outcome.

The following table gives the association of different variables with Hyperglycaemia.

Table 14: Association of Mechanical ventilator with Hyperglycaemia.

Variables	Sub Category	Hyperglycaemia		Total	p-value
		No	Yes		
Mechanical Ventilator	No	159 (96.95%)	45 (37.82%)	204 (72.08%)	< 0.001 ^{C*}
	Yes	5 (3.05%)	74 (62.18%)	79 (27.92%)	

Abbreviation: C – Chi square test, * indicates statistical significance.

From Chi square test, it is observed that, there is significant association of mechanical ventilator, inotropes and outcome with Hyperglycaemia. Out of 79 patients who were on mechanical ventilation, 74 (62.18%) were hyperglycaemic.

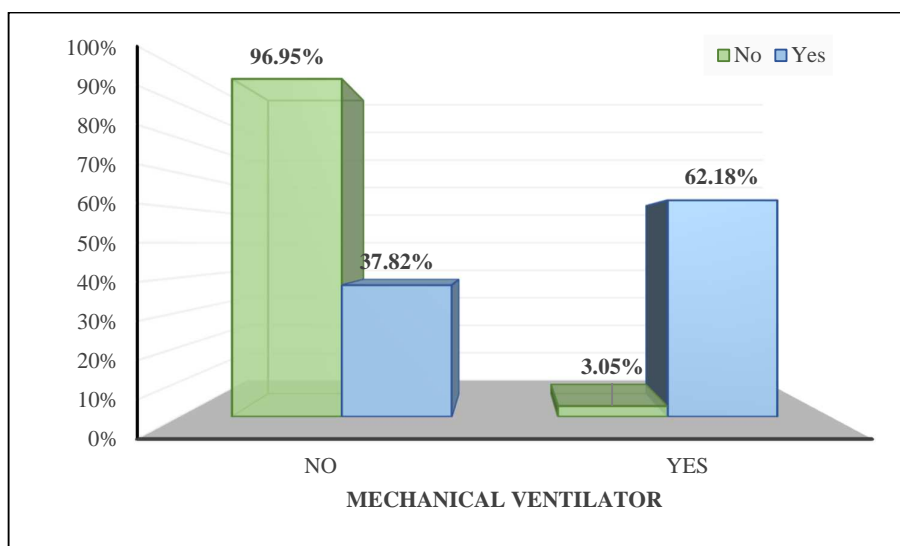


Figure 19: Distribution of subjects according to mechanical ventilator with hyperglycaemia.

Variables	Sub-Category	Hyperglycaemia		Total	p-Value
		No	Yes		
Inotropes	No	151 (91.52%)	39 (32.77%)	190 (66.9%)	<0.001 ^{C*}
	Yes	14 (8.48%)	80 (67.23%)	94 (33.1%)	

Abbreviation: C – Chi square test, * indicates statistical significance

Table 15- Association of inotropes with hyperglycemia.

From Chi square test, it is observed that, there is significant association of mechanical ventilator, inotropes and outcome with Hyperglycaemia. Out of 94 patients who required vasopressor support 80 patients were hyperglycaemic.

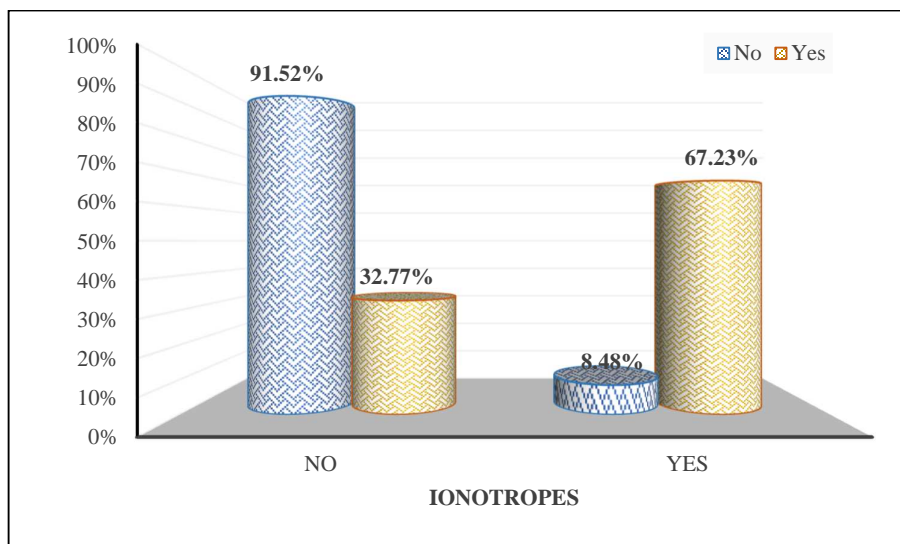


Figure 20: Distribution of subjects according to ionotropes with hyperglycaemia.

Variables	Sub-Category	Hyperglycaemia		Total	p-Value
		No	Yes		
Outcome	Not-survived	3 (1.82%)	69 (57.98%)	72 (25.35%)	<0.001 ^{C*}
	Survived	162 (98.18%)	50 (42.02%)	212 (74.65%)	

Abbreviation: C – Chi square test, * indicates statistical significance

Table 16- Distribution of subjects according to outcome with hyperglycemia.

From Chi square test, it is observed that, there is significant association of mechanical ventilator, inotropes and outcome with Hyperglycaemia. 72 patients did not survive, out of the, 69 were hyperglycaemic.

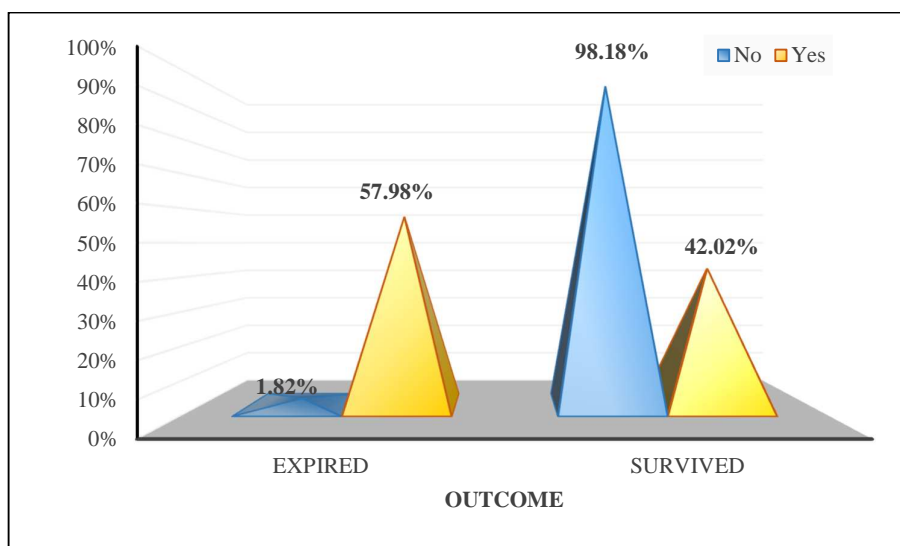


Figure 21: Distribution of subjects according to outcome with hyperglycaemia.

Table 17– Distribution of subjects according to PRISM Score on day 1 and day 4 with hyperglycemia.

HYPERGLYCEMIA	PRISM		OUTCOME
	DAY 1	DAY 4	
126-150mg/dl (total- 41)	High	Low	Survived-33 non-survived-8
>150 mg/dl (total-17)	High	Low	Survived-13 Non-survived-4
>200mg/dl (total-50)	High	High	Survived-4 non-survived-46

The following table gives the comparison of PRISM score over hyperglycaemia.

Table 18: Comparison of PRISM score over hyperglycaemia.

Hyperglycaemia	PRISM score		p-value
	Mean \pm SD	Median (Min, Max)	
No	5.58 \pm 6.12	5 (1, 74)	< 0.001 ^{MW*}
Yes	7.34 \pm 5.86	7 (0.29, 34)	

Abbreviation: MW – Mann Whitney U test, * indicates statistical significance.

From Mann Whitney U test, it is observed that, there is significant difference in the distribution of PRISM score over hyperglycaemia. Further, it can be observed that the PRISM score is higher among the subjects with hyperglycaemia.

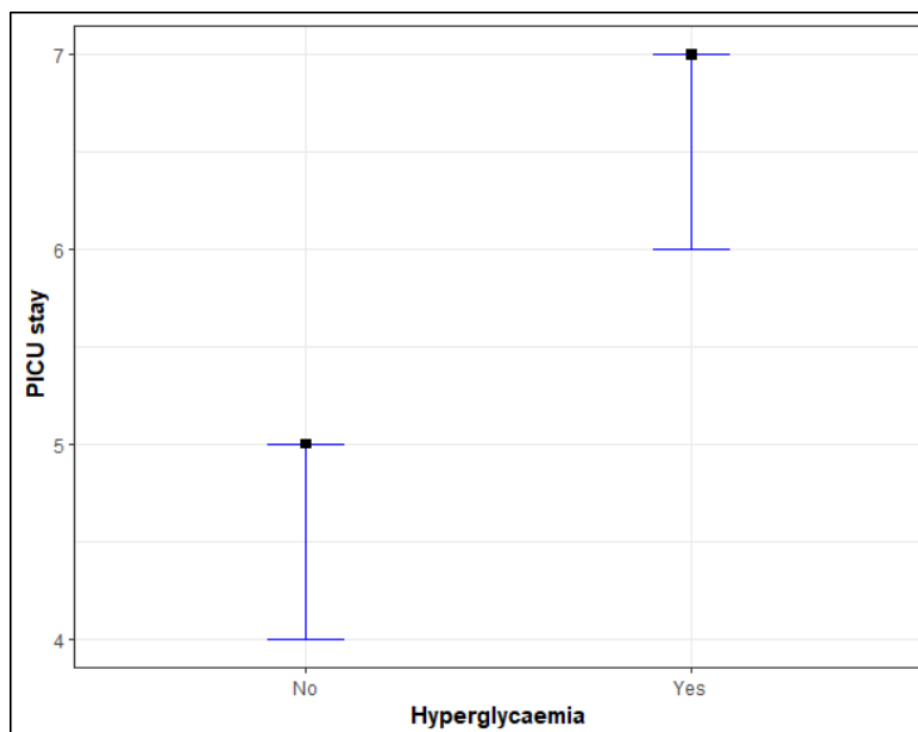


Figure 22: Median plot of PRISM score over hyperglycaemia.



DISCUSSION

Discussion: -

Hyperglycaemia is a prevalent condition among the critically ill children admitted in PICU. Our study done in 284 subjects found out that 41.9% have hyperglycaemia, 0.7% have hypoglycaemia and 57.39% have euglycaemi levels.

Among the hyperglycaemic, 42.86% that is 51 patients have glucose level more than 200mg/dl, 31.93% that is 38 patients have glucose level more than 126 mg/dl and 25.21% that are 30 patients have blood glucose level more than 200mg/dl. Similarly noted in a study done by Gurudutt Joshi *et al* (91) where the prevalence of hyperglycaemia was 80.3%, 72%, 31% when cut off level of blood glucose was considered >126mg/dl, >150mg/dl, >200mg/dl respectively. There is another study done by Ruiz Margo *et al* (92) reported that 50% of 353 patients had initial blood glucose level >120mg/dl. Faustino *et al* (93) also indicated that hyperglycaemia was prevalent in 70.4%, 44.5% and 22.3% in patients with blood glucose levels >120mg/dl, 150mg/dl and >200 mg/dl respectively.

There have been several mechanisms which lead to hyperglycaemia in critically sick child such as cytokine production, endothelial dysfunction, acute or transient dyslipidaemia, elevated gluconeogenesis, insulin resistance, production of hormones like epinephrine, norepinephrine, glucagon, growth hormone, and cortisol. All these further leading to metabolic disturbances, hyper coagulation and increased cellular apoptosis.

In this study, we found that there were 86 subject that is 30.28% with the age 1 month to 12 months, 131 subjects that is 46.13% with the age of 1 year to 10 years of age and 67 patients with 23.59 % with more than 10 years of age. There are other demographic characteristics also which are seen in this study, 41.9% (119 subjects) female patients and 58.1% (165 subjects) male patients.

This study also observed that there is a statistical significant (p -value- 0.0125) association between glycaemic levels with age. The prevalence of hyperglycaemia among study subjects in age group 1 to 12 months is 45 cases (52.33%), with 1 to 10 years is 42 cases (32.06%), and 32 cases with (47.76%). whereas there are 41 cases (47.67%), 87 cases (66.41%), and 35 cases (52.24%) with age from 1 to 12 months, 1 to 10 years, and >10 years respectively. In the hypoglycaemic range only 2 cases has been seen within 1 to 10 years of age (1.53%).

Chi- square test showed that on comparing morbidity and mortality with hyperglycaemia a statistical significant relation ($p < 0.001$) has been found with mechanical ventilation. Also reported in a study done by Gurudutt *et al*, mechanical ventilation was significantly higher for hyperglycaemia ($Z=6.77$; $p < 0.05$) (91). Similarly also noted in a study done by Michael Yung *et al* (94).

In our study out of 94 patients, 80 (67.23%) were hyperglycaemic requiring vasopressors. Similarly was found in 27 (73.0%) patients required vasopressor in the study done by Gurudutt Joshi *et al* (91), on comparing morbidity and mortality with hyperglycaemia, a statistical significant relation ($p < 0.05$) was found with mechanical ventilation requirement, vasopressor support requirement and mortality, prolonged duration of ventilation and increased vasopressor support was required with prolonged hyperglycaemia, also similar correlation was observed in Patki *et al*. (97)

In our present study Chi- square test showed that there are significant association between hyperglycaemia and outcome, mortality in PICU ($p < 0.001$) Out of 284 patients, hyperglycaemia was found in 119 (41.9%) patients, out of these 119 patients, 69 (57.98%) expired; whereas 50 (42.02%) survived. Similarly noted in study conducted by Gurudutt Joshi *et al*. (91). Ruiz Magro *et al*, also reported that the initial blood glucose levels were significantly higher in patients who died (92). Faustino and Apkon (93) also demonstrated that

hyperglycaemia is linked with greater in hospital mortality rate. On the contrast Edward Vincent et al (99), reported no association between initial blood glucose level and risk of death.

Various studies have also showed significant association between hyperglycaemia and type of disease. Brancho et al. (95) reported association between mortality and hyperglycaemia with septic shock, burns, traumatic brain injury, post cardiac surgery, and trauma. Chiaretti et al. (96) found association between severe sepsis and traumatic brain injury with hyperglycaemic mortality. Whereas Gurudutt et al (91) found no significant association between specific disease and hyperglycaemia. In our study we noted relationship between sepsis, trauma and respiratory infections with hyperglycaemic mortality.

It's also noted that Children with hyperglycaemia was reported to stay for longer duration in ICU by Palacio A et al (99). Faustino and Apkon (93) demonstrated that hyperglycaemia is correlated with longer duration of PICU stay.

STRENGTHS

- 1) There was no selection bias noted in our study, number of patients who were included in the study were specified.
- 2) It is a study done prospectively in a critically ill child.

LIMITATIONS

- 1) There is no definite criteria for diagnosing hyperglycaemia among patients without diabetes mellitus. In our study, we have divided the levels of hyperglycaemia, the problem here is in determining which blood glucose level is the most predictive of the outcome.
- 2) It is possible that hyperglycaemia and outcome may have a correlation, it is possible that hyperglycaemia and risk or mortality are both correlated by a third factor that is severity of illness.
- 3) The study was done in a single institution in Karnataka, whose population and disease spectrum can be different from other pediatric institutions.
- 4) Our study did not tell whether treatment of hyperglycaemia will improve the outcome of these patients.

We believe that hyperglycaemia in stress is a transient state or response to that acute illness, which do not require medical intervention.

- 5) The observational design cannot prove the causation, but can only demonstrate the association.



CONCLUSIONS

CONCLUSION

- Prevalence of mean hyperglycaemia in this study was 31.93%, 25.21% and 42.86% when cut off levels of hyperglycaemia was considered as >126mg/dl, >150 mg/dl and >200mg/dl respectively.
- There is a significant association between hyperglycaemia and morbidities like increased need for mechanical ventilation, vasopressors and prolonged PICU stay.
- Greater number of mortality is also seen in critically ill patients with hyperglycaemia.
- There is an association between hyperglycaemia and mortality in those patients who suffered from severe sepsis, trauma and respiratory infections.
- Our study was limited to find out the prevalence and assessment of associations, further studies are needed to evaluate whether the outcome can be improved by intervention like insulin therapy and better glucose control can be achieved.
- There is also a need for critical analysis for cut off levels of hyperglycaemia.



SUMMARY

SUMMARY

- This study was conducted in KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belagavi from Jan 2021 to Dec 2021. 284 children with hyperglycaemia who are critically ill and admitted in PICU between the ages of 1 month to 18 years were enrolled for the study after meeting the inclusion criteria.
- The primary goal was find the prevalence of hyperglycaemia in critically ill children, admitted in PICU.
- The secondary goal was to assess the clinical outcomes among hyperglycaemic based on PICU stay, inotropes used, mechanical ventilation and final outcome i.e., discharged healthy or death.
- When the patients were admitted in PICU, PRISM Scoring was also done. PRISM score is an indicator of morbidity and mortality. In our study, PRISM score was done on day 1 and day 4. It was observed that severe hyperglycaemic patients had higher PRISM score and hence higher mortality rate.
- Whereas on the other side, it was noted that PRISM score was lower in the hyperglycaemic with blood glucose of 126-150 mg/dl. And those patients still in hyperglycaemic range but had a lower PRISM score and hence low morbidity seen.

- Male to female ratio was 1.39:1 with 58.1% were males and 41.9% were females.
- Out of 284 patients, we got 86 patients (30.2%) in the age of 1 to 12 months of age, 131 (46.13%) patients between 1 to 10 years and patients above 10 years of age were 67 (23.59%).
- When we distributed out 284 subjects cases as per the glycaemic levels, out of these subjects, 163 patients (57.39%) have Euglycaemia, 119 patients (41.9%) have hyperglycaemia and 2 patients (0.7%) have hypoglycaemia.
- Euglycaemics is considered with when blood glucose is in range of 41-125 mg/dl, hypoglycaemia is considered with blood glucose level of <40 mg/dl, and hyperglycaemia is further cut off as >200 mg/dl, >150mg/dl, >126mg/dl.
- Total of 119 subjects had hyperglycaemia among 284, 42.86% have > 200mg/dl, 31.93% have >126mg/dl and 25.21% have >150mg/dl.
- In this study, we also found out the association of glycaemic levels according to age. Between the age of 1 to 12 months, euglycaemics were 41 cases that is 47.67, 45 cases of hyperglycaemia with 52.33% and no cases of hypoglycaemia.
- Between 1 to 10 years of age, we got 87 cases of euglycaemia that is 66.41%, 2 cases of hypoglycaemia with 1.% and 42 cases of hyperglycaemia with 32.06%. and in children above 10 years of age 35 cases were euglycaemics that is 52.24%, 32 cases are hyperglycaemics that is 47.76% and zero cases of hypoglycaemia.

- The prevalence of hyperglycaemia among the subjects in age group 1month-12 months is 52.33%, in age group 1year - 10 years is 32.06% and in age group above 10 years is 47.76%.
- The mean length of PICU was taken on the basis of all 284 patients stay. It was seen that the maximum length was upto 74 days and the minimum stay was upto 7 hours in the PICU. The length of PICU stay ranges from 7 hours to 74 days with mean length of PICU stay of 6.31 ± 6.07 days.
- The motive of our study was also to show the correlation between system involvements of the patient admitted in ICU to hyperglycaemia.
- It was seen that Out of 284 subjects, 88 patients had sepsis and hyperglycaemia that comes up to 30.9%, 20.42% patients had respiratory issues and hyperglycaemia that are 58 patients, 17.9% had central nervous system involvement and hyperglycaemia that are 51 patients, 44 patients had cardiac problems admitted in PICU developed stress hyperglycaemia that are 15.5%, 7.7% were of gastrointestinal involvement and hyperglycaemia (22 patients), and 7 cases of trauma which developed hyperglycaemia that is 2.6%.
- Our secondary motive was also to assess requirement of mechanical ventilator and inotropes requirement in hyperglycaemia as compared to euglycaemics or hypoglycaemic. Out of 284 subjects, 79 patients (27.82%) required mechanical ventilator and 94 patients (33.1%) required vasopressors. Out of 284 subjects, 74.65% survived whereas 25.35% did not survive.

- From Chi square test, it is observed that, there is significant association of mechanical ventilator, inotropes and outcome with Hyperglycaemia. Out of 79 patients who were on mechanical ventilation, 74 (62.18%) were hyperglycaemic.
- From Chi square test, it is observed that, there is significant association of mechanical ventilator, inotropes and outcome with Hyperglycaemia. Out of 94 patients who required vasopressor support 80 patients were hyperglycaemic.
- There is a significant association also noted between the outcome of patient and hyperglycaemia.
- From Chi square test, it is observed that, there is significant association of mechanical ventilator, inotropes and outcome with Hyperglycaemia. 72 patients did not survive, out of the, 69 were hyperglycaemic.
- Hyperglycemia was also assessed in association with PRISM scoring, From Mann Whitney U test, it is observed that, there is significant difference in the distribution of PRISM score over hyperglycaemia. Further, it can be observed that the PRISM score is higher among the subjects with hyperglycaemia.



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ANNEXURE I – CONSENT FORM

INFORMED CONSENT FOR PARTICIPATION IN RESEARCH

TITLE OF RESEARCH STUDY:

“PREVALENCE AND CLINICAL OUTCOME OF HYPERGLYCEMIA IN CRITICALLY ILL CHILDREN- ONE YEAR CROSS SECTIONAL STUDY.”

Principal Investigator :

REG NO.BM0120019

Post graduate student,
department of pediatrics –
Jawahar Lal Nehru Medical college,
belagavi-590010

Guide:

DR.

PROFESSOR

DEPARTMENT OF PEDIATRICS,
KLE ACADEMY OF HIGHER
EDUCATION & RESEARCH
JAWAHARLAL NEHRU MEDICAL
COLLEGE, BELGAUM -590010.

You are hereby requested to involve your child in the above said research to be conducted at KLE'S Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum from Jan 2021 to December 2021 by me.

INTRODUCTION & PURPOSE OF THE STUDY

The study aims to know the prevalence and clinical outcome of hyperglycemia in critically ill children.

Voluntary participation

Your child's participation in this study is your voluntary decision. Whether to participate or not to participate will not affect your current or future relationship with the KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum.

You are free to discontinue the participation in the study at any time for any reasons and you will not be paid any reimbursement for participation in the research.

Risk and benefits

There are no major risks involved, other than discomfort and pain caused during collection of biological sample. No study related injuries are expected in this research.

10

Privacy and Confidentiality

The only people who will know that you are a research participant are member of the research team. No information about you or provided by you, during research will be disclosed to others without your written consent. When the results of the research are published or discussed in the conferences, no information will be disclosed that would reveal your identity. Any information obtained in connections with this study and that can be identified with you remain confidential and will be disclosed only with your permission.

Queries

If you have any queries you may contact

REG NO.BM0120019

Post Graduate Student

Department of Pediatrics

JNMC, Belagavi-590010

PROFESSOR

DEPARTMENT OF PEDIATRICS,

KLE ACADEMY OF HIGHER EDUCATION & RESEARCH,

JAWAHARLAL NEHRU MEDICAL COLLEGE,

BELGAUM -590010.

If you have any questions about your rights or research participation you may contact

Chairman ethical committee:

DR. HARSHA HEGDE,

CHAIRPERSON, JNMC, IEC AND SCIENTIST D, ICMR, NATIONAL INSTITUTE OF TRADITIONAL
MEDICINE.

KLE ACADEMY OF HIGHER EDUCATION & RESEARCH.

JAWAHARLAL NEHRU MEDICAL COLLEGE,

BELGAUM -590010.

STATEMENT OF CONSENT

I hereby voluntarily agree for my child's participation in this study. I understand that even if I choose to allow my child to take part in this study I have the liberty to withdraw at any time. My signature below indicates that I have read or have been told about this entire consent form including the risks and benefits and have had all my questions answered. I will be given a copy of this consent form.

Signature of the patient: _____

Date: _____

Name: _____

Signature of the authorized representative/ parent: _____

Date: _____

Name: _____

Relation to the Subject: _____

Signature of the witness: _____

Date: _____

Name: _____

Signature of investigator: _____

Date: _____

Name: _____

ANNEXURE II- PROFORMA

Proforma-

- 1)IP NO.-
- 2)DATE-
- 3)SERIAL NO.-

SOCIO-DEMOGRAPHIC DETAILS-

INFORMANT-

- 1)NAME-
- 2)RELATION-
- 3)PROFESSION-
- 4)EDUCATION-
- 5)ADDRESS-
- 6)TELEPHONE-

PATIENT-

- 1)NAME-
- 2)AGE-
- 3)SEX-
- 4)DOA-
- 5)WEIGHT-
- 6) **Initial Impression-**

Consciousness- Alert/irritable/unresponsive/uncnscious

Breathing- increased WOB/decreased WOB/absent efforts/abnormal sounds

Colour- pallor/mottling/cyanosis

7) **Primary Assessment-**

Airway-clear/maintainable/not maintainable

Breathing- Respiratory rate-

Respiratory efforts- nasal flaring/retractions/head bobbing

Chest expansions and distal air movements

Lung and airway sounds- crackles/stridor/wheeze/grunt

Spo2-

Circulation- HR- rhythm-
Pulses- peripheral - central-
CFT- BP-
Skin colour-

Disability- level of consciousness by AVPU scale-
Pupil – PERRL

Exposure- trauma/bleeding/burns

8) Secondary assessment-

Focussed history- Signs and symptoms
Allergy
Medications
Past medical history
Last meal
Events leading to current illness

Focussed examination- head to toe examination-

Respiratory system-

Central nervous system-

Per abdomen-

Cardiovascular system-

Investigations-

Prism III score	On admission	Fourth day	score
Systolic bp			
Diastolic bp			
Heart rate			
Respiratory rate			
Pao ₂ /Fio ₂			
Paco ₂			
GCS			
Pupillary reaction			
PT/Aptt			
Total bilirubin			
Potassium			
Calcium			
Glucose			
Bicarbonate			

Glucose measurement-

DAY 1 - on admission -

12th hourly-

≥126 mg/dl	<126 mg/dl
6 th hourly till they become <126mg/dl	12 th hourly masurement

Diagnosis-

Treatment given-

Final outcome-

Picu stay-

ANNEXURE III – PRISM SCORING

Table 1: PRISM III score

Variables	Age restrictions and Range		Score
Systolic blood pressure in mm Hg	Infants	Children	
	130-160	50-200	2
	55-65	65-75	
	> 160	>200	6
	40-54	50-64	
	< 40	<50	7
Diastolic blood pressure in mm Hg	All ages		6
Heart rate in beats per minute	Infants	Children	
	> 160	> 150	4
	<90	< 80	4
Respiratory rate in breaths per minute	Infants	Children	
	61-90	51-70	1
	>90	> 70	5
	apnea	apnea	5
PaO ₂ /FiO ₂	All ages	200-300	2
		<200	3
PaCO ₂ in torr (mm Hg)	All ages	51-65	1
		>65	5
Glasgow coma score	All ages	<8	6
Pupillary reactions	All ages	Unequal or dilated	4
		Fixed and dilated	10
PT/PTT	All ages	1.5 times control	2
Total bilirubin mg/dL	>1 month	> 3.5	6
Potassium in mEq/L	All ages	3.0-3.5	1
		6.5-7.5	1
		< 3.0	5
		> 7.5	5
Calcium in mg/dL	All ages	7.0-8.0	2
		12.0-15.0	2
		<7.0	6
		>15.0	6
Glucose in mg/dL	all ages	40-60	4
		250-400	4
		<40	8
		>400	8
Bicarbonate in mEq/L	all ages	<16	3
		>32	3

AGE	GENDER	DATE OF ADMISSION	WEIGHT	DIAGNOSIS	SYSTEM AFFECTED	OXYGEN SUPPLEMENTATION AND DURATION	MECHANICAL VENTILATOR	IV FLUIDS AND DURATION	IONOTROPES AND DURATION	ANTIBIOTICS	OTHER TREATMENTS	PICU STAY	GLYCAEMIC LEVELS ON ADMISSION	BLOOD GLUCOSE	PRISM score	PROBABILITY OF MORTALITY/MORBIDITY	OUTCOME
1 year	female	01-01-2021	8.8 kg	A/H/O RTA (Road Traffic Accident)	Trauma	4 days by nasal prongs, hfnc for 9 days	no	13 days	adrenaline infusion x 2 days	20 days	inj tranexa x2 days	15 days	160mg/dl	hyperglycaemia	19	81%	survived
2 years	female	02-01-2021	10 kg	anaphylactic reaction to food allergen associated with respiratory and circulatory collapse	RESPIRATORY	1 day by mask	no	1 day	-	3 days	adrenaline inj, adrenaline nebulizations	3 days	124mg/dl	euglycaemia	15	48%	survived
12 year	female	03-01-2021	50kg	AML 3RD CLAD/ARAC CYCLE WITH FEBRILE NEUTROPENIA WITH SEPTIC SHOCK	sepsis	-	48 hours	6 days	-	6 days	inj cytrabin, cladrabine	6 days	200mg/dl	hyperglycaemia	28	99%	expired
4 months	male	04-01-2021	6 kg	Lower respiratory tract infection	RESPIRATORY	by hood x 3 days	-	5 days	-	5 days	nebulizations	5 days	125mg/dl	euglycaemia	9	16%	survived
3yr 6 month	female	04-01-2021	12 kg	Lower respiratory tract infection	RESPIRATORY	3 days by hood	no	5 days	-	5 days	nebulizations	5 days	125mg/dl	euglycaemia	6	4%	survived
9 year	Male	05-01-2021	45kg	GB syndrome with tracheostomy tube insitu	CNS	24 hrs by mask	24 hours	3 days	-	14 days	nebulizations, antiepileptics	14 days	184mg/dl	hyperglycaemia	8	16%	survived
11 years	Male	10-01-2021	37Kg	Snake Bite	sepsis	-	no	5days	dobutamine infusion x 2 days	5days	Inj Vitamin K, anti snake venom	6days	158mg/dl	hyperglycaemia	11	16%	survived
3 Year	female	10-01-2021	11kg	Case of Diamond blackfan syndrome	sepsis	-	no	-	-	9days	FFP,RDP transfusion, nasal packing	9 days	100mg/dl	euglycaemia	13	16%	survived
48days	Male	15-01-2021	3 kg	septic arthritis of left knee	sepsis	-	no	-	-	7 days	pa killers	8 days	114mg/dl	euglycaemia	5	4%	survived
5 years	Male	15-01-2021	18kg	severe Dengue fever without warning signs	sepsis	-	no	3days	-	5 days	emset, pantop	5 days	101mg/dl	euglycaemia	3	4%	survived
15 years	Male	15-01-2021	44.5 kg	nephrotic syndrome, AKI secondary to SBP, mild hydronephrosis	GIT	2 days by mask	no	5 days	-	5 days	ICD insertion, tab ommacortil, antihypertensive	5 days	126mg/dl	hyperglycaemia	7	16%	survived
4 months	female	15-01-2021	5kg	Thalassemia	GIT	1 day by nasal prongs	no	1 days	-	-	PCU transfusion x 1 setting	1 day	78mg/dl	euglycaemia	12	16%	survived
12months	female	17-01-2021	6kg	OP/C/O wilms tumor with bronchopneumonia	RESPIRATORY	3 days HFNC	no	6 days	-	7days	Nebulizations, inj mgso4	7 days	123mg/dl	euglycaemia	16	48%	survived
8yr 6 month	Male	18-01-2021	40 kg	epileptic encephalopathy, myoclonic epilepsy with LRTI	CNS	-	no	3days	-	5 days	Inj sodium valproate	5 days	117mg/dl	euglycaemia	10	16%	survived
8yr 6th month	Male	18-01-2021	35Kg	acyanotic CHD-Ventricular Septal Defect with bronchopneumonia in CCF	CVS	6 days with CPAP	no	6 days	dobutamine infusion x 4 days	10days	decongestive drugs, nebulization	10days	93mg/dl	euglycaemia	9	16%	survived
14 year	Male	19-01-2021	51kg	New Onset Refractory Status Epilepticus (NORSE) ?paraneoplastic encephalitis	CNS	-	10days	10days	adrenaline x 6 days, noradrenaline x 5 days	10days	Ketamine infusion, antiepileptics	10days	276mg/dl	hyperglycaemia	41	99%	Expired
2 years	Male	20-01-2021	8kg	Bronchiolitis	RESPIRATORY	2 days with nasal prongs	no	3 days	-	7days	aminophylline, Nebulizations, inj hydrocortisone	5days	101mg/dl	euglycaemia	13	16%	survived
7months	Male	20-01-2021	6.8kg	infantile tremor syndrome ith acynotic congenital heart disease with VSD with CCF with LRTI	CVS	2 days by mask	no	7days	dobutamine infusion x 72 hrs	7 days	decongestive drugs, Nebulizations	9 days	120mg/dl	euglycaemia	16	48%	survived
5months	female	21-01-2021	3.8 kg	seborrheic dermatitis with LRTI with acute GE with staphylococcal scalded skin syndrome	RESPIRATORY	-	no	4 days	-	10 days	syp pct, syp yal, syp zincogut	10days	128mg/dl	hyperglycaemia	8	16%	survived
5yrs 6month	Male	24-01-2021	16kg	FIRES (febrile infection related epilepsy syndrome)	CNS	-	5days	18days	nor adrenalnex 2 days	31days	ketogenic diet, topiramate, parampanel	32day	152mg/dl	hyperglycaemia	19	48%	survived
4 years	Male	25-01-2021	12Kg	Acute gastroenteritis with moderate dehydration	GIT	-	no	2days	-	5days	ORS, zinc, emset	4days	95mg/dl	euglycaemia	5	4%	survived
2 months	male	27-01-2021	5 kg	septic arthritis	sepsis	-	no	1 day	-	8 days	PCT, painkillers	8 days	105mg/dl	euglycaemia	9	10%	survived
5years	Male	29-01-2021	15kg	Bicuspid Aortic valve underwent Balloon valvotomy	CVS	-	no	1 day	-	5days	procedure- aortic balloon valvotomy	4 days	93mg/dl	euglycaemia	8	16%	survived
13years	Male	02-02-2021	60kg	OP/C/O Appendicitis	sepsis	-	no	10days	-	10days	procedure, painkillers	10days	124mg/dl	euglycaemia	5	4%	survived
13 Years	female	03-02-2021	8 kg	benign familial infantile seizures with aspiration pneumonia with severe respiratory distress	RESPIRATORY	2 days by nasal prongs	14 hours	3 days	adrenaline infusion 10 hours	3days	hydrocortisone,antiepileptics,nebulizations	3days	140mg/dl	hyperglycaemia	22	95%	expired
14 years	male	13-02-2021	65kg	idiopathic thrombocytopenic pupura	sepsis	-	no	5 days	-	5 days	methy prednisolone, corticosteroids x 6weeks	5 days	115mg/dl	euglycaemia	5	4%	survived
2years	Male	03-02-2021	12kg	case of tuberous sclerosis with rhabdomyosarcoma with sepsis	sepsis	-	4days	4days	adrenalnex 4 days, nor adrenaline x 3 days	4days	antifungal,antiepileptics	4days	292mg/dl	hyperglycaemia	40	99%	expired
10years	Male	04-02-2021	25kg	Mirror traumatic Brain injury due to fall from terrace (7Feet)	CNS	-	no	2days	-	5days	supportive care	4days	119mg/dl	euglycaemia	9	16%	survived
12year	Male	04-02-2021	35kg	case of T-All with febrile netropenia	sepsis	-	no	-	-	5days	filgastrim, inj PCT	4days	114mg/dl	euglycaemia	5	4%	survived
3months	Male	07-02-2021	4kg	laryngomalacia with LRTI	RESPIRATORY	3days by nasal prongs	no	3 days	-	5days	Nebulizations, positioning, domestal	6days	118mg/dl	euglycaemia	7	16%	survived
11year	Male	08-02-2021	25kg	chronic Kidney Disease stage V-D secondary to CAKUT	GUT	-	no	8days	-	5days	hameodialysis, calcium supplementation,PCV transfusion	8days	124mg/dl	euglycaemia	12	16%	survived
45days	Male	08-02-2021	1.85 kg	laryngomalacia with LRTI with GERD	RESPIRATORY	-	no	24 hours	-	7 days	Nebulizations- levosalbutamol, positioning	7 days	96mg/dl	euglycaemia	6	4%	survived
2yr 6months	female	11-02-2021	10kg	Bronchiolitis	RESPIRATORY	-	no	-	-	5days	Nebulizations	7days	93mg/dl	euglycaemia	5	4%	survived
4months	female	12-02-2021	3.8kg	recurrent bronchopneumoniae with lower lobe collapse of left side with lasrge ASD with severe PAH with streptococcal sepsis	sepsis	9days by nasal prongs, HFNC 1 day	3days	13days	dobutamine x 4 days, adrenaline x 3 days	13days	decongestives, Nebulizations	13days	273mg/dl	hyperglycaemia	29	99%	expired
6year	female	13-02-2021	15kg	Severe dengue fever	GIT	-	no	4 days	-	5days	emset, pantop	5days	111mg/dl	euglycaemia	5	4%	survived
1yr 2 months	Male	14-02-2021	9.5kg	febrile seizures	CNS	-	no	2 days	-	5 days	Inj Lorazepam, tab frisium	2 days	137 mg/dl	hyperglycaemia	5	4%	survived
3months	Male	14-02-2021	5kg	fissure in right lower lobe due to previous ICD insertion with severe bronchopneumoniae with failure to thrive	RESPIRATORY	-	2days	2 days	adrenaline x 2 days , noradrenaline x 2 days	2 days	-	2days	190mg/dl	hyperglycaemia	33	99%	expired
7months	Male	14-02-2021	6kg	Foreign Body Aspiration of (hair clip)	septic shock	-	1day	1 day	adrenaline x 1 day	1 day	open thoracotomy	1days	260mg/dl	hyperglycaemia	32	99%	expired
5 months	Female	16-02-2021	5kg.	Thalassaemia Major	HEMATOLOGY	24 hours by mask	no	1 day	-	-	PCV transfusion	1 day	78mg/dl	euglycaemia	7	4%	survived
3years	female	16-02-2021	10kg	Case of Diamond blackfan syndrome with nasal bleeding	ONCOLOGY	-	no	-	-	5days	Nasal Packing, FFP,RDP transfusion, botrocloct drops	9days	100mg/dl	euglycaemia	13	16%	survived
45days	female	17-02-2021	2kg	Thalassaemia major	HEMATOLOGY	1day by mask	no	1day	-	-	PCV transfusion	1days	78mg/dl	euglycaemia	12	16%	survived
16years	female	20-02-2021	31kg	Organo phosphorus Poisoning	sepsis	-	2days	8days	-	8 days	atropine, PAM, Nebulizations	8days	121mg/dl	euglycaemia	23	95%	survived
4 years	male	21-02-2021	15 kg	right sided empyema	sepsis	by mask 2 days	no	7 days	-	12 days	ICD insertion, PCT	10 days	135mg/dl	hyperglycaemia	6	4%	survived
2yr 6months	female	22-06-2021	10kg	Dilated cardiomyopathy with sever left ventricular dysfunction in CCF	CVS	hfnc x 1 day	12 hrs	1 day	adrenaline x 24 hours	1 day	decongestives	1day	182mg/dl	hyperglycaemia	20	81%	expired
6 months	Male	24-02-2021	4.2kg	Cyanotic CHD- tricuspid atresia with large ASD with large VSD with tiny PDA with severe PS	CVS	8 days by nasal prongs	no	6 days	dobutamine infusion 48 hrs	7 days	tab propanolol	8days	126mg/dl	hyperglycaemia	10	16%	survived
15yrs	Male	01-03-2021	44kg	left temporal lobe abcess with mass effect with left temporal craniotomy	CNS	-	no	-	-	11days	anti epileptic	11days	100mg/dl	euglycaemia	9	16%	survived
4months	Male	01-03-2021	4kg	congenital muscular dystrophy with walker warburg syndrome	CNS	-	8 days	9 days	adrenaline infusion x 6 days	9 days	antiepileptics, physiotherapy	9 days	126mg/dl	hyperglycaemia	31	99%	expired
16years	Male	01-03-2021	48kg	Known case of Haemophila B with Intracranial bleed	HEMATOLOGY	-	12hrs	12hrs	adrenaline x 10 hours	1 day	FFP, RDP transfusion	1day	184mg/dl	hyperglycaemia	27	95%	expired
5yrs	Male	02-03-2021	16kg	severe Traumatic Brain Injury with signs of raised ICT with SAH and SDH with diffuse axonal injury	trauma	-	no	8days	-	8 days	anti epileptic, inj mannitol	8days	244mg/dl	hyperglycaemia	12	16%	survived
11 months	female	04-03-2021	7 kg	acute gastroenteritis with severe dehydration	GIT	-	no	2 days	-	5 days	ORS, zinc, emset	3 days	97 mg/dl	euglycaemia	8	4%	survived
8years	female	05-03-2021	35kg	K/C/O Dravet syndrome with severe Bronchopneumoniae with covid RTPCR Positive in status epilepticus	CVS	12 days by mask	5 days	17 days	adrenaline x 3days, noradrenaline x 3 days	17 days	aminophylline infusion, ketamine infusion, tranexa	17 days	231mg/dl	hyperglycaemia	30	99%	expired
7months	female	06-03-2021	6.5kg	Infantile dengue fever with warning signs with right sided pleural effusion- mild	RESPIRATORY	-	no	4days	dopamine x 2 days	7 days	inj lasix	4days	107mg/dl	euglycaemia	13	48%	survived
1 year 2 months	female	07-03-2021	10 kg	ileocecal intussusception underwent USG guided hydrostatic reduction	GIT	-	no	2 days	-	5 days	PCV transfusion, procedure	2 days	98 mg/dl	euglycaemia	9	16%	survived
2 months	female	08-03-2021	3 kg	operated case of biliary atresia with umbilical hernia with hepatopulmonary syndrome with cystic biliary atresia	GIT	-	9 days	9 days	adrenaline x 7 days , noradrenaline x 9 days	9 days	hepatic drip, kasai portoenterostomy operation	9 days	169mg/dl	hyperglycaemia	41	99%	expired
7 months	Male	10-03-2021	6kg	large perimembranous VSD in CCF with failure to thrive	CVS	-	3 days	10 days	ubtamine infusion 3 days, adrenaline infusion x 2 da	10 days	decongestive therapy	10 days	116mg/dl	euglycaemia	19	81%	expired
3 months	female	11-03-2021	7 kg	supraventricular tachycardia (atrial fibrillations)	CVS	by nasal prongs x 1 day	5 hours	1 day	-	1 day	vagal maneuver,adenosine, DC shock, metoprolol	1 day	311mg/dl	hyperglycaemia	20	81%	Expired
13yrs	Female	11-03-2021	45kg	GB Syndrome	CNS	-	no	3 days	-	5 days	-	6days	92mg/dl	euglycaemia	8	4%	survived
1yr 6 months	female	11-03-2021	4kg	ARDS secondary to septicemia with pericardial effusion with massive pneumothorax with ICD in situ	sepsis	HFNC 8 days	no	8 days	dobutamine infusionx 5 days	8 days	ICD insertion, tab ommacortil,antihypertensive	12days	120mg/dl	euglycaemia	6	4%	survived
4years	Male	11-03-2021	12kg	Acute Gastroenteritis with moderate dehydration	GIT	-	no	2 days	-	5days	ORS, zinc, emset	4days	95mg/dl	euglycaemia	5	4%	survived
5yrs	female	13-03-2021	12.7kg.	OP/C/O VP shunt Insertion with chiari malformation with ? Aqueuductal stenosis	CNS	-	no	10 days	-	15 days	diamox tab	15days	122mg/dl	euglycaemia	5	4%	survived
3years	Male	13-03-2021	10kg	Bronchopneumonia	RESPIRATORY	10hrs by mask	no	16hrs	-	5days	Nebulizations	4days	109mg/dl	euglycaemia	10	16%	survived
5 years	female	18-03-2021	14 kg	subdural hematoma in left frontal region with frontal bone fracture	CNS	-	no	2 days	-	5 days	mannitol, emset, pantop, antiepileptics	2 days	119mg/dl	euglycaemia	5	4%	survived
11 months	female	19-03-2021	8.4kg	acyanotic CHD with large fossa ovalis ASD without PAH without CCF with severe LRTI	CVS	5 days by nasal prongs	no	8 days	-	7 days	nebulization, decongestives, inj lasix	8 days	126mg/dl	hyperglycaemia	7	16%	survived
8months	Male	22-03-2021	7kg	left torsion testis with bilateral undescended testis underwent left orchietomy with right orchidopexy	GUT	-	no	3 days	-	7 days	procedure, painkillers, PCT inj	3 days	129mg/dl	hyperglycaemia	10	16%	survived
8 year	male	27-03-2021	24 kg	case of second degree burn with compartment syndrome underwent fasciotomy	sepsis	-	no	4 days	-	15 days	fasciotomy, hydroheal ointment, nadibact	4 days	129mg/dl	hyperglycaemia	13	48%	survived
3years	Male	28-03-2021	10kg	WALRI (wheeze associated lower respiratory tract infection)	RESPIRATORY	2days by nasal prongs	no	2 days	-	5days	Nebulizations, inj dexa	4days	109mg/dl	euglycaemia	9	16%	survived
4 years	Male	02-															

11months	male	07-05-2021	4.5kg	hemolytic uremic syndrome caused by E. coli associated with anaemia, hypocalcaemia with septic shock	sepsis	o2 by mask 24 hours, 3days by HFNC	7days	11days	Nor adrenaline x 8 days	11days	Nephroprotective antibiotics, lasix, calcium, PCV, FFP transfusion	11days	240mg/dl	hyperglycaemia	43	99%	expired	
18months	male	08-05-2021	5kg	Viral pneumoniae	RESPIRATORY	-	no	1day	-	3days	nebulization, inj dexa	3days	91mg/dl	euglycaemia	5	4%	survived	
2months15days	Male	10-05-2021	5kg	Congenital acyanotic heart disease, ventricular septal defect with left to right shunt with bronchopneumoniae no PAH, no CCF	CVS	-	no	3 days	-	5days	decongestives, nebulizations	6days	100mg/dl	euglycaemia	5	4%	survived	
14years	female	10-05-2021	30kg	Enteric fever with intestinal ulcers	sepsis	-	no	8days	-	14days	pantoprazole, inj PCT, painkillers, PCU transfusion x 1 setting	8days	130mg/dl	hyperglycaemia	10	16%	survived	
5years	female	12-05-2021	14kg	WALRI (wheeze associated lower respiratory tract infection)	RESPIRATORY	3 days by mask	no	3 days	-	7 days	asthalin and budesort nebulization	5 days	113mg/dl	euglycaemia	7	16%	survived	
4 years	male	13-05-2021	17 kg	asthma	RESPIRATORY	-	no	1 day	-	5 days	low dose inhaled corticosteroids, nebulizations, montelukast tab	5 days	111mg/dl	euglycaemia	16	48%	survived	
10years	female	15-05-2021	24kg	MISC-myocarditis with acute gastroenteritis	CVS	-	30hours	3days	dobutamine x 12 hours, adrenaline x 24 hours	2days	-	-	-	132mg/dl	hyperglycaemia	16	48%	expired
4 years	female	15-05-2021	13kg	A/H/O RTA on 9/5/21 with multiple fractures with subarachnoid bleed Bilaterally	trauma	-	7 days	17 days	naline x 4 days, noradrenaline x 3 days, dopamine x 2	24 days	FFP & PCV transfusion, tranexa	25days	156mg/dl	hyperglycaemia	20	81%	survived	
5years	male	17-05-2021	12kg	Dengue shock syndrome	sepsis	3days by Mask	no	9days	dobutamine x 2 days, adrenaline x 3 days	9days	RDP transfusion, haemacel	9days	113mg/dl	euglycaemia	5	4%	survived	
2years	female	18-05-2021	6kg	Acute gastroenteritis with some dehydration	GIT	-	no	1day	-	3days	ORS, zinc, emset	2days	93mg/dl	euglycaemia	5	4%	survived	
12years	female	19-05-2021	36kg	A/H/O consumption of All-Out (prallethrin poisoning) leading to with pulmonary oedema, altered sensorium, convulsions, with hypersensitivity syndrome	CNS	-	3days	3days	dopamine infusion 2days	3days	Antihypertensives, gastric lavage	3days	390mg/dl	hyperglycaemia	39	99%	expired	
5 years	male	19-05-2021	20 kg	hemophilia B	sepsis	-	no	1 day	-	5 days	FFP, RICE protocol, PCT	3 days	97mg/dl	euglycaemia	13	16%	survived	
2months26days	female	20-05-2021	4.5kg	Staphylococcal scalded skin syndrome (Ritter disease) ?PID	sepsis	-	no	6days	-	14days	dressing with silver sulphadiazine	6days	95mg/dl	euglycaemia	13	48%	survived	
40 days	male	26-05-2021	3.4kg	Non Paroxysmal repetitive seizures	CNS	-	no	3 days	-	3 days	eptoin inj, pyrofer, gardenal	3 days	115mg/dl	euglycaemia	8	16%	survived	
1 year	Male	26-05-2021	4kg	C/o VACTERL with OP/C/Of Tracheo-oesophageal fistula with single kidney with LRTI	RESPIRATORY	2days by hood	no	2days	-	5 days	Nebulizations	5 days	112mg/dl	euglycaemia	7	10%	survived	
3 months	Male	28-05-2021	5.4kg	Bronchiolitis	RESPIRATORY	48HOURS by nasal prongs	no	48 hours	-	7 days	Nebulizations	6 days	100mg/dl	euglycaemia	6	4%	survived	
5yrs 1month	Male	28-05-2021	12.4kg	bronchiolitis	RESPIRATORY	2days by mask	no	2 days	-	5 days	Nebulizations	5days	98mg/dl	euglycaemia	8	15%	survived	
1 year	male	29-05-2021	4.5kg	Kawasaki disease	HEMATOLOGY	-	no	1day	-	7days	IVIG, low dose aspirin	5days	128mg/dl	hyperglycaemia	16	48%	survived	
14years	male	30-05-2021	28kg	Thalassaemia in CCF	HEMATOLOGY	-	no	3 days	-	3days	PCV transfusion, decongestives, lasix inj	3 days	112mg/dl	euglycaemia	7	16%	survived	
7yrs	Male	01-06-2021	22kg	Portal cavernoma with portal Hypertension with oesophageal & rectal varices	GIT	-	no	3 days	-	10 days	octreotide infusion, tranexa	3 days	121mg/dl	euglycaemia	2	4%	survived	
6 months	female	01-06-2021	4.3kg	K/C/O Tetralogy of fallot with cardiogenic shock	CVS	-	24 hours	24 hours	adrenaline and dobutamine x 24 hours	24 hours	decongestive treatment	1 day	240mg/dl	hyperglycaemia	39	99%	expired	
6 year	male	03-06-2021	9 kg	PSGN	sepsis	by mask 2 days	no	4 days	-	7 days	salt restriction, water restriction, lasix, antihypertensives, calcium	12 days	99mg/dl	euglycaemia	12	48%	survived	
11 years	male	06-06-2021	17 kg	coronavirus associated MISC with MODS	sepsis	-	4 days	4 days	dobutamine x 4 days	4 days	IVIG, methylprednisolone, tocilizumab	4 days	395mg/dl	hyperglycaemia	39	99%	expired	
4 years	Male	07-06-2021	12kg	k/C/O diamond blackfan syndrome	ONCOLOGY	-	no	-	-	7 days	FFP transfusion, botroclot drops, nasal packing	9 days	100 mg/dl	euglycaemia	12	16%	survived	
6 year	Male	07-06-2021	16kg	Bronchiolitis with Bronchopneumia	RESPIRATORY	5 days by mask	no	3 days	-	7 days	Nebulizations, inj dexa	5 days	150mg/dl	hyperglycaemia	7	4%	survived	
9 months	Male	07-06-2021	4 kg	staphylococcal scalded skin syndrome with toxic shock syndrome with cerebral palsy with rickettsia	sepsis	-	no	7 days	-	7 days	antiepileptics, Nebulizations, syp baclofen	7 days	150mg/dl	hyperglycaemia	8	16%	survived	
10 years	Male	08-06-2021	24kg	Acute Osteomyelitis of Right hip and femur and dengue fever in recovery phase	sepsis	-	no	3 days	-	14 days	painkillers	5days	97mg/dl	euglycaemia	8	16%	survived	
4years	female	09-06-2021	18 kg	tubercular meningitis with abducens nerve involvement	sepsis	HFNC x2 days	12 days	14 days	nor adrenaline x 8 days	14 days	ATT, steroids	14 days	290mg/dl	hyperglycaemia	34	99%	survived	
13 years	Male	10-06-2021	30kg	acute Promyelocytic leukemia with CNS positive cytogenetics with bacterial meningitis	sepsis	3 days by mask	4days	7 days	adrenaline infusion x 6 days	7 days	RDP, PCV transfusions, mannitol and 3%NS infusion	7days	218mg/dl	hyperglycaemia	23	95%	expired	
11years	Male	11-06-2021	15kg	K/C/O Hypertrophic cardiomyopathy with Branchopneumoniae	CVS	-	5days	11 days	milrinone 3 days	11 days	tab carvedilol, inj metoprolol, Nebulizations	11days	100mg/dl	euglycaemia	11	16%	survived	
5 year	male	12-08-2021	20 kg	asthma category I	RESPIRATORY	-	no	-	-	5 days	low dose inhaled corticosteroids,	5 days	118mg/dl	euglycaemia	15	48%	survived	
10 years	male	13-06-2021	22kg	acute necrotizing encephalopathy	CNS	1 day by mask	4 days	5 days	dopamine x 4 days	5 days	tocilizumab, perampamel, antiepileptics,	5 days	310 mg/dl	hyperglycaemia	50	99%	expired	
2 year 6 months	female	15-06-2021	11kg	DCM with severe left ventricular dysfunction in CCF	CVS	-	24 hours	1 day	adrenaline 24 hrs	1 day	decongestives	1 day	182mg/dl	hyperglycaemia	20	81%	expired	
14 year	male	17-06-2021	52 kg	henoch schonlein purpura	HEMATOLOGY	-	no	1 day	-	5 days	methylprednisolone, Nsaids, antihypertensives, PCT	5 days	120mg/dl	euglycaemia	12	16%	survived	
5 year 6months	Male	18-06-2021	15kg	FIRES (febrile infection related epilepsy syndrome)	CNS	-	5days	18days	nor adrenaline infusion x 2 days	15days	ketogenic diet, topiramate, perampamel	31 days	152mg/dl	hyperglycaemia	18	48%	survived	
13years	Male	28-06-2021	40kg	Snake bite(haematotoxic)	sepsis	-	no	6 days	dobutamine x 48 hours	6 days	inj vit k, anti snake evemon	6 days	158mg/dl	hyperglycaemia	11	16%	survived	
4 years	male	28-06-2021	16 kg	community acired pneumoniae (atypical bacterial pneumoniae)	RESPIRATORY	by mask x 2 days	no	2 days	-	5 days	PCT, nsaids, nebulizations	5 days	106mg/dl	euglycaemia	5	4%	survived	
7years	Male	29-06-2021	21kg	acute gastroenteritis with moderate dehydration	GIT	-	no	2 days	-	7 days	zinc, ORS	3days	82mg/dl	euglycaemia	9	16%	survived	
2months	female	29-06-2021	3.9kg	Non-Paroxysmal repetitive seizures	CNS	-	no	3 days	-	3 days	antiepileptics, vit B6	3 days	154mg/dl	hyperglycaemia	8	4%	survived	
1 year 2 months	male	30-06-2021	6 kg	viral laryngotracheobronchiis	RESPIRATORY	by nasal prongs x 4 days	no	4 days	-	5 days	adrenaline nebulizations, inj dexa, budesonide nebulization	4 days	98mg/dl	euglycaemia	11	16%	survived	
3 months	female	30-06-2021	4 kg	K/C/O VSD with LRTI	CVS	4 days by nasal prongs,	no	5 days	-	10 days	decongestive therapy, Nebulizations 10 days	6 days	138mg/dl	hyperglycaemia	6	4%	survived	
9yrs 2months	Male	01-07-2021	15kg	T- lymphoblastic lymphoma with transverse sinus thrombosis (left side)	ONCOLOGY	-	no	2 days	-	5days	low molecular weight heparin, antiepileptics	4 days	123mg/dl	euglycaemia	5	4%	survived	
14yrs	Male	01-07-2021	58kg	severe dengue with dengue shock syndrome with myocarditis	sepsis	-	7 days	7 days	dopamin, adrenaline x 7 days, dobutamine 5 days	7 days	aspirin, prednisolone,	7days	250mg/dl	hyperglycaemia	36	99%	expired	
14yrs	female	01-07-2021	45kg	C/o Rheumatic heart disease with LRTI	CVS	5 days by mask	no	5 days	-	5 days	methy, steroids	5 days	129mg/dl	hyperglycaemia	8	16%	survived	
6years	female	02-07-2021	12kg	severe dengue with warning signs with Right sided pleural effusion with left side hematoma neck	sepsis	4 days by nasal prongs	2 days	10days	dopamine x 3 days, adrenaline x 4 days	10days	RDP transfusion, lasix infusion	10days	160mg/dl	hyperglycaemia	20	81%	expired	
8 years	female	02-07-2021	21kg	Rickettsial brain stem encephalitis with positive RGA score	sepsis	2 days by mask	6days	8 days	adrenaline x 4 days	8days	antiepileptics	8days	240mg/dl	hyperglycaemia	34	99%	expired	
4years	female	02-07-2021	10kg	case of Pulmonary atresia with VSD with DORV with cyanotic spells in CCF with failure to thrive	CVS	2days by nasal prongs	4 days	6days	dobutamine x 6 days, adrenaline x 5 days,	6 days	inj metoprolol, ketamine infusion, decongestives	6days	241mg/dl	hyperglycaemia	18	81%	expired	
6years	Male	03-07-2021	15kg	viral fever	RESPIRATORY	-	no	-	-	5 days	-	1 day	112mg/dl	euglycaemia	5	4%	survived	
10months	female	03-07-2021	4.9kg	foreign body aspiration	RESPIRATORY	2 hours by hood	no	5 hours	-	5 days	-	2 days	112mg/dl	euglycaemia	5	4%	survived	
16years	Male	03-07-2021	55kg	Severe dengue with warning signs	sepsis	6 hours by mask	no	4 days	dopamine x 29 hours,	7 days	emset, pantop	7days	135mg/dl	hyperglycaemia	8	16%	survived	
10years	Male	11-07-2021	22kg	dengue like illness & K/C/O VSD since 2 years of age	sepsis	-	no	6 days	-	6 days	-	6days	119mg/dl	euglycaemia	3	4%	survived	
3months	Male	03-07-2021	7kg	ionotrope refractory shock with acute gastroenteritis ? Inorn error of metabolism	sepsis	-	7hrs.	7 hours	adrenaline, dobutamine x 7 hours	7 hrs	-	7 hrs	240mg/dl	hyperglycaemia	32	99%	expired	
10months	Male	04-07-2021	5.5kg	Cyanotic CHD- total anomalous pulmonary venous connection in CCF (cardiac type) with recurrent cynotic spells	CVS	8 days by nasal prongs	9hrs	8 days	dobutamine x 8 days, adrenaline x 9 hours	8 days	ketamine infusion, cardioprotective treatment, inj metoprolol	8days	142mg/dl	hyperglycaemia	25	95%	expired	
1 year 7 months	male	04-07-2021	12 kg	simple febrile seizures	CNS	-	no	12 hours	-	3 days	frisium tab, paracetamol,	3 days	114mg/dl	euglycaemia	8	16%	survived	
8 years	female	05-07-2021	13kg.	dystonic quadriplegic CP secondary to birth asphyxia with microcephaly with failure to thrive with GDD	CNS	-	no	-	-	5 days	lacosamide, syp brevil, cap neuro D3, tab torpanel	5days	122mg/dl	euglycaemia	5	4%	survived	
11years	Male	05-07-2021	22.6kg	K/C/O fanconi's Anaemia with C/O Bleeding from gums	HEMATOLOGY	-	no	2 days	-	3 days	xamic, RDP transfusion, inj vit k	3days	122mg/dl	euglycaemia	6	4%	survived	
8 months	female	05-07-2021	4kg.	Bronchopneumonia	RESPIRATORY	HFNC x 12 hours , 2 days by nasal prongs	no	5 days	-	6 days	Nebulizations	6days	118mg/dl	euglycaemia	13	16%	survived	
9 months	Male	05-07-2021	10kg	Acute Tracheo bronchitis- croup	RESPIRATORY	2 days by nasal prongs	no	1 day	-	3 days	adrenaline nebulizations	4days	110mg/dl	euglycaemia	10	16%	survived	
7 years	female	05-07-2021	26kg	SLE with hepatic encephalopathy(stage 1)	CNS	3 days by hfnc	no	-	-	21 days	immunomodulators, antiepileptics	21days	114mg/dl	euglycaemia	14	48%	survived	
15years	Male	06-07-2021	35kg	serum sickness in a known case of thalassaemia major	sepsis	-	no	-	-	5 days	methy prednisolone, naproxen, avil	5days	110mg/dl	euglycaemia	8	16%	survived	
15months	Male	07-07-2021	4.5kg	simple febrile seizures	CNS	-	no	-	-	3days	frisium tab, paracetamol,	2 days	126 mg/dl	euglycaemia	4	4%	survived	
3years	female	07-07-2021	8 kg	Dhatura Poisoning	sepsis	3 days by mask	no	3 days	-	7days	inj neostigmine	7days	115mg/dl	euglycaemia	15	48%	survived	
14 years	female	07-07-2021	9 kg.	severe dengue with dengue shock syndrome with B/L pleural effusion with worm infestation with CP	sepsis	2 days by nasal prongs, HFNC X 3 days	no	9days	dobutamine infusionx 2days	16days	lasix infusion, antiepileptic, deworming tab	10days	120mg/dl	euglycaemia	10	16%	survived	
4 years	female	08-07-2021	10kg	Febrile seizures	CNS	-	no	12hrs	-	3 days	frisium tab, paracetamol,	4days	121mg/dl	euglycaemia	6	4%	survived	
14years	female	09-07-2021	28kg	Thalassaemia in CCF with tooth abscess.	HEMATOLOGY	-	no	-	-	-	pcv transfusion, decongestive therapy	3days	112mg/dl	euglycaemia	7	16%	survived	
14years	Male	10-07-2021	39kg	posterior reversible encephalopathy syndrome secondary to focal segmental glomerulosclerosis	CNS	-	no	3 days	-	-	labetalol infusion, amlodipine, envas	5 days	95mg/dl	euglycaemia	5	4%	survived	
5years	female	10-07-2021	16.5kg	WALRI (wheeze associated lower respiratory tract infection)	RESPIRATORY	4days by hood	no	2 days	-	10days	Nebulizations, inj hydrocortisone	7 days	210mg/dl	hyperglycaemia	2	4%	survived	
8 months	Male	10-07-2021	4.5kg	Bronchopneumonia	RESPIRATORY	HFNC 1 day, by Nasal Prongs 2 days	no	5 days	-	6 days	Nebulizations	6days	118mg/dl	euglycaemia	13	16%	survived	
6 years	female	11-07-2021	22kg	K/C/O Nephrotic Sydrome with spontaneous bacterial peritonitis with right sided mild pleural effusion	GUT	48hrs by mask	no	4 days	-	10days	lasix infusion 3 days	7 days	129mg/dl	hyperglycaemia	9	16%	survived	
1 month	Male	13-07-2021	3.5kg.	acyanotic CHD, VSD with left to right shunt, with CCF, with FTT, with covid status positive with LRTI	CVS	72hrs by mask, HFNCx 90 hours	24 hours	9days	dobutamine x 90 hours, adrenaline x 30 hours	7days	decongestive therapy, inj vit k	9 days	190mg/dl	hyperglycaemia	18	81%	expired	
9months	Male	15-07-2021	5.1kg	SMA with aspiration	CNS	-	10 days	10 days	adrenaline infusion 2 days	13 days	Nebulizations	13 days	135mg/dl	hyperglycaemia	40	99%	expired	
8 years	Male	15-07-2021	40kg	Epileptic encephalopathy? progressive myoclonic epilepsy, ?dravet's syndrome	CNS	2 days by mask	3 days	6 days	arenaline x 3 days	6 days	lacosamide, syp brevil, cap neuro D3, tab torpanel	6 days	130mg/dl	hyperglycaemia	18	81%	expired	
8yrs 5months	Male	20-07-2021	24kg	Epileptic encephalopathy (myoclonic epilepsy) with LRTI	CNS	-	no	4days	-	7 days	inj sodium valproate	5 days	117mg/dl	euglycaemia	10	16%	survived	
14yrs	female	23-07-2021	48.5kg	dengue fever in critical phase	sepsis	-	no	5 days	-	-	pantop, emset	5days	120mg/dl	euglycaemia	13	48%	survived	
16yrs	female	23-07-2021	51kg	GB syndrome	CNS	CPAP x 2 days	no	5 days										

9year	female	04-09-2021	21kg	Enteric fever	sepsis	-	no	1 day	-	7 days	pct	4days	119mg/dl	euglycaemia	7	16%	survived
4 years	female	07-09-2021	12 kg	community acired pneumoniae (atypical bacterial pneumoniae)	sepsis	by mask 2 days	no	-	-	5 days	serrapeptidases, pct,nebulizations	6 days	106mg/dl	euglycaemia	8	16%	survived
14years	Male	10-09-2021	36kg	PRES	CNS	-	no	2days	-	-	labetalol tab, envastab, amlodipine tab, lasix tab	6days	143mg/dl	hyperglycaemia	13	48%	survived
6 year	male	10-09-2021	18kg	leptospirosis meningoccephalitis	sepsis	HFNC x 1 day	4 days	6 days	noradrenaline 4 days	6 days	antiepileptics	6 days	211mg/dl	hyperglycaemia	30	99%	expired
2 years	male	11-09-2021	8kg	enteric fever with acute gastroenteritis with moderate dehydration	GIT	-	no	3 days	-	5 days	-	5 days	106mg/dl	euglycaemia	17	48%	survived
7 years	female	11-09-2021	30 kg	post covid lung syndrome	CVS	HFNC x 2 days	no	3 days	-	7 days	dexamethasone	6 days	79mg/dl	euglycaemia	5	4%	survived
12 years	female	12-09-2021	39kg	acute necrotizing encephalitis	CNS	by mask x 2 days	4 days	6 days	dopamine x 4 days	6 days	tocilizumab, perampanel, antiepileptics,	6 days	310mg/dl	hyperglycaemia	44	99%	expired
6yrs	Male	13-09-2021	19kg	Acute exacerbation of Asthma category I	RESPIRATORY	12hrs by Mask	no	1day	-	3 days	low dose inhaled corticosteroids	1day	121mg/dl	euglycaemia	6	4%	survived
12 years	male	13-09-2021	44kg	infectious mononucleosis a case of burkitts lymphoma	ONCOLOGY	by mask x 4 days	no	8 days	nor adrenaline x 4 days	8 days	antiviral, prednisolone	8 days	260mg/dl	hyperglycaemia	36	99%	expired
2 year	female	14-09-2021	5 kg	unilateral acute otitis media of right side	sepsis	-	no	1 day	-	14 days	nsaids	3 days	87mg/dl	euglycaemia	6	4%	survived
6 months	Male	15-09-2021	5kg	Bronchiolitis with operated case of ARM (stage II)	RESPIRATORY	3 days by nasal prongs	no	5days	-	7days	nebulizations	8days	121mg/dl	euglycaemia	14	48%	survived
2year3 months	female	16-09-2021	7 kg	DCM with severe LV dysfunction in CCF	CVS	-	1 day	1 day	-	1 day	dobutamine x 12 hours	1 day	188mg/dl	hyperglycaemia	20	81%	expired
9 year	female	16-09-2021	45 kg	hepatitis -b	sepsis	-	no	3 days	-	-	-	4 days	69mg/dl	euglycaemia	16	48%	survived
12 years	male	16-09-2021	45kg	enteric fever	sepsis	-	no	1 day	-	7 days	-	3 days	119mg/dl	euglycaemia	5	4%	survived
13 years	male	17-09-2021	47 kg	autoimmune encephalitis	CNS	-	10 days	10 days	adrenaline x 5 days, noradrenaline x 4 days	10 days	adrenaline	10 days	400mg/dl	hyperglycaemia	28	99%	expired
4 years	female	17-09-2021	14kg	viral fever with bronchopneumoniae	RESPIRATORY	2 days	no	1 day	-	5 days	nebulizations	1 day	133mg/dl	hyperglycaemia	6	4%	survived
6 years	male	19-09-2021	18 kg	post streptococcal glomerulonephritis with bilateral pleural effusion	sepsis	by mask x 2 days	no	6 days	-	10 days	salt restriction, water restriction, lasix,antihypertensives, calcium	16 days	99 mg/dl	euglycaemia	14	48%	survived
2 months	female	19-09-2021	5 kg	coarctation of aorta with severe PAH with LRTI underwent balloon dilatation	CVS	by nasal prongs x 5 days	no	5 days	dobutamine 3 days	7 days	sildenafil, decongestives	10 days	131mg/dl	hyperglycaemia	15	16%	survived
3 year	female	21-09-2021	14kg	kawasaki disease	HEMATOLOGY	-	no	1 day	-	5 days	IVIG, aspirin, inj PCT	4 days	125mg/dl	euglycaemia	10	16%	survived
9 months	Male	30-09-2021	4kg	dengue fever	sepsis	-	no	-	-	3 days	-	4days	91mg/dl	euglycaemia	7	15%	survived
7months	Male	01-10-2021	6.5kg	febrile seizures	CNS	-	no	-	-	-	-	3days	82mg/dl	euglycaemia	6	4%	survived
2years	Male	01-10-2021	13kg	refractory cardiac failure secondary to severe metabolic acidosis ?inborn error of metabolism ? Organic acidemia	sepsis	12days nasal prongs	12hrs	12 days	adrenaline x 24 hours	12 days	-	12 days	310mg/dl	hyperglycaemia	34	97%	expired
15years	Male	02-10-2021	53kg	Left sided Pneumothorax after getting hit by a football	RESPIRATORY	-	no	5days	-	5days	ICD insertion	4days	121mg/dl	euglycaemia	9	16%	survived
11years	Male	02-10-2021	55kg	dengue fever in critical phase with dengue hepatitis with MISC myocarditis	GIT	HFNC x 5days	3 days	11days	mine x 5 days, adrenaline x 10 days, dobutamine 2	11 days	IVIG, heptoprotective drugs	11days	173mg/dl	hyperglycaemia	22	95%	expired
2yrs 6months	Male	02-10-2021	8kg	congenital heart disease VSD with severe PS in CCF	CVS	-	no	10 days	dobutamine x 4 days	12 days	cardioprotective drugs	12days	120mg/dl	euglycaemia	17	48%	survived
8months	Male	02-10-2021	4.5kg	sepsis induced HUS with right sided empyema with burkholderia sepsis with candida sepsis	sepsis	5 days by mask	no	10 days	-	10 days	PCV transfusion, ICD insertion	10days	129mg/dl	hyperglycaemia	13	48%	survived
1yr 5months	female	02-10-2021	4kg	late onset hemorrhagic disease of newborn	CNS	-	24hrs	2 days	adrenaline 24 hours	2 days	inj vit k	2days	36mg/dl	hypoglycaemia	41	99%	expired
7 years	male	03-10-2021	33kg	acute pharyngitis with Melsacc score of 4	RESPIRATORY	-	no	4 day	-	10 days	Nsaids	5 days	106mg/dl	euglycaemia	9	16%	survived
6months	Male	05-10-2021	5kg	Bronchiolitis with Bronchopneumonia	RESPIRATORY	-	no	4days	-	8days	Nebulizations	8days	108mg/dl	euglycaemia	13	48%	survived
16years	female	06-10-2021	30kg	organophosphorus poisoning	sepsis	-	no	6 days	-	8days	atropine, pralidoime, nebulizations	8days	121 mg/dl	euglycaemia	13	48%	survived
2yr 6months	female	10-10-2021	7kg	k/C/O diamond blackfan syndrome	ONCOLOGY	-	no	8 days	-	7 days	FFP, RDP transfusion	9days	100mg/dl	euglycaemia	12	16%	survived
13years	male	10-10-2021	60kg	OP/C/O Appendicitis	sepsis	-	no	10days	-	10days	procedure, painkillers	10days	124mg/dl	euglycaemia	5	4%	survived
6yr 6month	female	10-10-2021	14kg	complex febrile seizures	CNS	6 hours by nasal prongs	no	12 hours	-	-	frisium tab, paracetamol,	1day	121mg/dl	euglycaemia	7	16%	survived
4 years	Male	11-10-2021	10kg	Portal cavernoma with portal Hypertension with oesophageal & rectal varices	GIT	-	no	3days	-	5days	tranexa,octreotide infusion	3days	121mg/dl	euglycaemia	2	4%	survived
14 years	Male	11-10-2021	65kg	idiopathic thrombocytopenic pupura	HEMATOLOGY	-	no	4days	-	5days	corticosteroids x 6 weeks	5days	115mg/dl	euglycaemia	5	4%	survived
2years	Male	11-10-2021	12kg	case of tuberous sclerosis with cardiac rhabdomyosarcoma with candidal sepsis	CVS	-	4days	4 days	adrenaline x 4 days, dobutamine x 3 days	4 days	antiepileptics	4days	292mg/dl	hyperglycaemia	40	99%	expired
1yr6month	Male	12-10-2021	10kg	K/C/O Hypertrophic cardiomyopathy with bronchopneumonia	CVS	-	5days	11days	milrinone 3 days	11 days	thiamine,tab metoprolol, carvedilol, nebulizations	11days	100mg/dl	euglycaemia	11	16%	survived
10years	Male	12-10-2021	25kg	Minor traumatic Brain Injury due to fall from terrace	trauma	-	no	2days	-	5days	pain killers, mannitol	4days	119mg/dl	euglycaemia	9	16%	survived
14years	Male	12-10-2021	42kg	Dengue fever in febrile phase	sepsis	-	no	3days	-	-	emet, pantop	3 days	100mg/dl	euglycaemia	5	4%	survived
16years	Male	13-10-2021	70kg	Brain death with hydrocephalous with raised ICT with tonsillar herniation secondary to fulminant cerebellitis	CNS	-	3days	3 days	adrenaline x 3 days	3days	mannitol, 3% NS	3days	160mg/dl	hyperglycaemia	25	95%	expired
12years	Male	16-10-2021	35kg	case of T-All with febrile neutropenia	sepsis	-	no	1 day	-	5days	filgastrim, inj PCT	4days	114mg/dl	euglycaemia	5	4%	survived
3months	Male	17-10-2021	4.5kg	Laryngomalacia with LRTI	RESPIRATORY	3days by nasal prongs	no	2 days	-	5days	domestal, position, nebulization	6days	118mg/dl	euglycaemia	7	16%	survived
2yrs 6months	female	17-10-2021	10kg	Bronchopneumonia	RESPIRATORY	-	no	-	-	5days	Nebulizations	1days	93mg/dl	euglycaemia	5	4%	survived
11 years	Male	18-10-2021	15kg	Chronic kidney disease stage V-D with Juvenile nephronophthisis with left kidney agenesis on Hemodialysis with anaemia & hypocalcemia	GUT	-	no	8days	-	8days	antihypertensive,PVC transfusion,calcium,hemodialysis	8days	124mg/dl	euglycaemia	12	16%	survived
6 years	female	18-10-2021	15kg	Severe dengue fever	sepsis	-	no	4days	-	5days	-	5days	111mg/dl	euglycaemia	5	4%	survived
4months	female	20-10-2021	4kg	Bronchopneumonia with left lower lobe collapse with large PDA with severe PAH with candida sepsis with scabies	RESPIRATORY	9days by nasal prongs, HFNC 1 day	3days	13days	adrenaline x 3 days, dobutamine 4 days,	13days	decongestives, antifungals,scabies t/Nebulizations	13days	273mg/dl	hyperglycaemia	29	99%	Expired
7months	male	21-10-2021	6kg	Foreign Body Aspiration thoracotomy done	RESPIRATORY	-	1 day	1day	adrenaline x 1 day	1day	procedure	1day	260mg/dl	hyperglycaemia	32	99%	Expired
1 year	Male	23-10-2021	12Kg	Inborn error of Metabolism with status epilepticus	sepsis	-	12hrs	12hrs	adrenaline infusion	-	antiepileptics	1day	299mg/dl	hyperglycaemia	31	97%	expired
5 months	male	03-11-2021	5 kg	cyanotic congenital heart disease, tetralogy of fallot in cyanotic spells without CCF	CVS	HFNC x 10 hours	2 days	2 days	-	2 days	ketamine, sodium bicarbonate,metoprolol,	2 days	210mg/dl	hyperglycaemia	25	95%	expired
3 years	female	07-11-2021	11 years	simple febrile seizures	CNS	-	no	1 day	-	3 days	frisium tab, paracetamol,	2 days	81mg/dl	euglycaemia	9	16%	survived
15 years	female	08-11-2021	40 kg	henoch schonlein purpura	HEMATOLOGY	-	no	2 days	-	5days	methyprednisolone(pulse therapy), Nsaids, PCT	5 days	128mg/dl	hyperglycaemia	12	16%	survived
19 months	male	08-11-2021	7.5 kg	Viral pneumoniae	RESPIRATORY	-	no	1 day	-	3 days	nebulization, inj dea	3 days	91mg/dl	euglycaemia	5	4%	survived
10 months	female	09-11-2021	9 kg	VSD with LRTI	CVS	by nasal prongs 4 days	no	4 days	-	5 days	nebulizations, decongestives	6 days	122mg/dl	euglycaemia	6	4%	survived
9 years	male	10-11-2021	22 kg	post viral myocarditis in a K/C/O hypertrophic obstructive cardiomyopathyin CCF with severe PAH	CVS	by mask 3 days	no	5 days	dobutamine x 2 days	5 days	thiamine, sildenafil,decongestives	5 days	121mg/dl	euglycaemia	11	16%	survived
3months	female	11-11-2021	6 kg	Lower respiratory tract infection	RESPIRATORY	by mask x 1 day	no	-	-	3 days	nebulizations	3 days	97mg/dl	euglycaemia	12	16%	survived
17 years	male	11-11-2021	75 kg	dengue fever	sepsis	-	no	-	-	3 days	emet,pantop	4 days	100mg/dl	euglycaemia	10	16%	survived
4 years	male	11-11-2021	16kg	hemophilia A moderate in severity	HEMATOLOGY	-	no	1 day	-	3 days	FFP,RICE protocol,PCT	3 days	103mg/dl	euglycaemia	11	16%	survived
10 years	female	14-11-2021	25 kg	protracted bacterial bronchitis	RESPIRATORY	-	no	-	-	2 weeks	nebulization	3 days	71mg/dl	euglycaemia	9	16%	survived
6months	Female	16-11-2021	6kg	Acyanotic congenital heart disease large ASD with left to right shunt with moderate to severe PAH in CCF	CVS	-	no	-	-	-	cardioprotective drugs	4days	110mg/dl	euglycaemia	9	16%	survived
12 year	female	17-11-2021	19kg	post covid19 syndrome	CVS	by mask x 24 hours	no	1 day	-	3 days	salbutamol nebulizations	3 days	101mg/dl	euglycaemia	9	16%	survived
2 months	female	18-11-2021	4 kg	acyanotic congenital heart disease, large VSD with severe PAH in CCF	CVS	HFNC 2days	no	2 days	-	7 days	decongestives, vsd patch closure	7 days	120mg/dl	euglycaemia	10	16%	survived
7 years	female	18-11-2021	20 kg	urosepsis	sepsis	-	no	1 day	-	7 days	-	74days	100mg/dl	euglycaemia	5	4%	survived
5 year	male	19-11-2021	18 kg	urticaria with angiedema	sepsis	-	no	1 day	-	5 days	adrenaline inj, prednisolone, citrizine	2 days	120mg/dl	euglycaemia	9	16%	survived
2 year	female	19-11-2021	7 kg	tubercular meningitis with basal meningial hyperdensities	sepsis	-	12 days	12 days	adrenaline x 10 days	12 days	ATT, prednisolone	12 days	230mg/dl	hyperglycaemia	31	99%	expired
2 year	female	22-11-2021	8 kg	bilateral acute otitis media	sepsis	-	no	10 days	-	10 days	painkillers, Nsaids	3 days	87mg/dl	euglycaemia	6	4%	survived
11months	female	24-11-2021	5kg	Hypertrophic Cardiomyopathy in CCF with LRTI with severe PPHN	CVS	-	6days	6days	dobutamine, adrenaline x 6 days	6 days	decongestive, thiamine inj	6days	244mg/dl	hyperglycaemia	20	81%	expired
45days	Male	25-11-2021	1850gms	Laryngomalacia with failure to thrive	RESPIRATORY	-	no	2 days	-	5 days	Nebulizations	4days	202mg/dl	hyperglycaemia	8	4%	survived
9 months	Male	30-11-2021	8kg	Dengue fever	sepsis	-	no	4days	-	3 days	-	4days	91mg/dl	euglycaemia	7	15%	survived
2months 2days	Male	30-11-2021	3kg	acyanotic CHD, large perimembranous VSD with large VSD wth LRTI with FTT	CVS	-	9 days	9 days	dobutamine 9 days , adrenaline x 5 days	9 days	decongestives	9days	414mg/dl	hyperglycaemia	10	95%	expired
13yrs	Female	30-11-2021	27kg	Hypertensive emergency secondary to oreganophophorus poisoning	sepsis	1 day by mask	6days	7days	dopamine x 1 day	7days	labetalol infusion, atropine,PAM	7days	162mg/dl	hyperglycaemia	31	99%	expired
7months	Female	01-12-2021	8kg	febrile seizures	sepsis	15hrs by nasal prongs	no	-	-	-	frisium tab, paracetamol,	4days	112mg/dl	euglycaemia	3	4%	survived
2yrs 6months	Male	01-12-2021	7kg	simple febrile seizures with LRTI	CNS	1day by nasal prongs	no	1day	-	3days	Nebulizations	3days	123mg/dl	euglycaemia	14	48%	survived
17yrs	Male	01-12-2021	51kg	case of Haemophilia -B with Intracranial Bleed	CNS	-	12hrs	12hrs	adrenaline x 12 hours	1 day	FFP,RDP transfusion	1day	184mg/dl	hyperglycaemia	27	95%	expired
8months	male	02-12-2021	14kg	Sepsis indeved HUS with septicemic shock	sepsis	-	8days	10 days	inone x 2 days, dobutaminox 2 days, adrenaline x 3	8days	-	10days	135mg/dl	hyperglycaemia	27	95%	survived
1 year 1 month	male	03-12-2021	7 kg	acute bacterial meningitis	sepsis	by nasal prongx 2 days	no	1 day	-	14 days	antiepileptics	4 days	130 mg/dl	hyperglycaemia	19	48%	survived
15months	female	03-12-2021	4.5kg	Haemorrhagic disease of Newborn	CNS	-	no	2 days	adrenaline 2 days	2 days	inj vit k	2days	36mg/dl	hypoglycaemia	36	99%	expired
4 years	female	03-12-2021	15 kg	dengue fever	sepsis	-	no	2 days	-	-	emet, pantop	3 days	102 mg/dl	euglycaemia	5	4%	survived
10months	Male	04-12-2021	7.7kg	Infantile Nephrotic syndrome	sepsis												