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"SERUM LACTATE LEVEL AS A PREDICTOR OF OUTCOME IN PATIENTS WITH SUSPECTED SEPSIS ADMITTED IN PEDIATRIC INTENSIVE CARE UNIT"

BY

Dr. SHUBHAM KUMAR

DISSERTATION SUBMITTED TO

B.L.D.E { DEEMED TO BE UNIVERSITY }

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In partial fulfillment of the requirements for the degree of

DOCTOR OF MEDICINE IN PEDIATRICS

UNDER THE GUIDANCE OF

Dr. S V PATIL

PROFESSOR

DEPARTMENT OF PEDIATRICS

BLDE[DU]

SHRI B.M. PATIL MEDICAL COLLEGE, HOSPITAL & RESEARCH CENTRE

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I hereby declare that this dissertation titled " **SERUM LACTATE LEVEL AS A PREDICTOR OF OUTCOME IN PATIENTS WITH SUSPECTED SEPSIS ADMITTED IN PEDIATRIC INTENSIVE CARE** " has been prepared by me under the supervision and guidance of Dr SV PATIL sir, Professor, Department of Pediatrics. This is being submitted to BLDE (Deemed to be University) Shri. B. M. Patil Medical College, Hospital & RC, Vijayapura, Karnataka in partial fulfilment of the requirement for award of master's degree in Pediatrics. This work has not been submitted to any University by me for the award of any degree.

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VIJAYAPURA

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Abbreviations

ATP	Adenosine triphosphate
IL2	Interleukin-2
HIC	High-income countries
MODS	Multiple organ dysfunction syndrome
MCT	Monocarboxylate transporter
LMIN	Lower-middle-income nations
LDH	Lactate dehydrogenase
PAMP	Pathogen-associated molecular patterns
SIRS	Systemic inflammatory response syndrome
SOFA	Sepsis Related Organ Failure Assessment

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"SERUM LACTATE LEVEL AS A PREDICTOR OF OUTCOME IN PATIENTS WITH SUSPECTED SEPSIS ADMITTED IN PEDIATRIC INTENSIVE CARE"

Introduction

Sepsis is the important causes of death among children in globally . Systemic inflammatory response syndrome in the presence of or as a result of a suspected or confirmed infection was considered sepsis; organ malfunction was considered severe sepsis; and cardiovascular dysfunction was considered septic shock. In children, septic shock and severe sepsis are the main causes of death.[1]. A dysregulated host response to infection causes sepsis, a potentially fatal clinical condition marked by immunological dysregulation, systemic inflammation, and microcirculatory abnormalities. The particular clinical characteristics are contingent upon the patient's position on the continuum, which spans from sepsis to multiple organ dysfunction syndrome (MODS) and septic shock.[2]

For non-cardiac intensive care unit patients most common cause is sepsis. Despite improvements in treatment and the availability of critical care, the mortality rate for septic shock is between 40 and 60 percent. Sepsis has a very high mortality and morbidity rate; at least one out of every four cases result in death. Sepsis can be confirmed by the presence of infection. SIRS criteria, which include symptoms including tachycardia, hypothermia, and leucocytosis, are the foundation for the precise confirmation of sepsis.[3]

According to data released in 2020, sepsis-related mortality accounted for 11 million deaths and 48.9 million cases globally, or 20% of all deaths. Children under the age of five accounted for

nearly half (20 million) of all estimated cases of sepsis worldwide.[4] An estimated 15 people out of every 1000 hospitalized patients will have sepsis as a side effect of their treatment. Although sepsis can strike anyone in the world, there are notable regional differences in incidence and death, with lower-middle-income nations (LMICs) having the highest rates [5].

In addition to harming the body's own tissues and organs, the reaction can result in shock, multiple organ failure, and occasionally even death if it is not identified and treated quickly.
Frequent symptoms of sepsis include fever, rapid breathing, elevated heart rate, disorientation, and bodily aches. Septic shock, multiple organ failure, and even death are possible outcomes.
Although bacterial infections typically induce sepsis, other infections including those caused by viruses, parasites, or fungus can also cause it. Medical attention, including the administration of intravenous fluids, antimicrobials, and other treatments, is necessary for its treatment. High lactate levels are a symptom of septic shock, one of the most dangerous medical situations. Even in wealthy nations, it adds to the mortality rate of 10%.[6]

In healthy people, serum lactate levels are typically modest. Only in diseases like lactic acidosis and hyperlactatemia are blood lactate levels visible. In hyperlactatemia they range from 2 to 5 mmol/l and above 5 mmol/l in lactic acidosis. Serum lactate levels in critically unwell individuals are therefore used as a diagnostic indicator. Anaerobic metabolism uses lactate as a byproduct. Hypoxia in the tissue is directly indicated by high lactate levels. An extended period of tissue hypoxia may harm the tissue irreparably and lead it to die. One common hallmark of patients with septic shock is organ failure. Hypoxia of the tissues is linked to organ failure. Long-term tissue hypoxia is linked to hyperlactatemia, which can lead to organ failure. Research on patient lactate correction has demonstrated that lowering lactate levels by alone does not enhance the prognosis of

patients in critical condition. Controlling morbidity and mortality would be much more effective with a combination therapy that improves oxygen supply while lowering lactate.[3]

Lactate levels may rise as a result of several circumstances that cause insufficient oxygen delivery, exaggerated oxygen demand, and reduced oxygen usage. A hallmark of sepsis and septic shock is hyperlactatemia. It is claimed that the mechanisms underlying hyperlactatemia in the two disorders differ. An elevated lactate level in sepsis indicates an enhanced glycolytic flux brought on by hypermetabolism; critically ill patients typically have blood lactate levels up to 18 mg/dl (2 mmol/l) as normal. The condition of Lactic acidosis occurs when lactate exceeds 45 mg/dl combined with pH values below 7.35 while hyperlactatemia exists when lactate ranges between 18 to 45 mg/dl without showing signs of metabolic acidosis. Elevated lactate serves as an early alert for a condition that may reverse whereas early septic shock suggests additional chances for quick treatment "there is still room".[7]

It is possible for critically ill patients to maintain normal blood pressure and urine output while in compensated septic shock. Early treatment depends on more than physiological measures because these patients require immediate intervention to survive. Among the biological assessment parameters for detecting hypoperfusion and measuring resuscitation results stands the plasma lactate value.

Early lactate clearance in critically ill people with septic shock can predict survival, and elevated lactate levels are associated with poor outcomes. Nevertheless, not enough research has been done on the prognostic value of lactate clearance in kids with severe sepsis.[6]

The survival rate dropped from 90% to 10% when human lactate levels rose from 2.1 to 8 mmol/l, according to groundbreaking research on the subject. Lactate levels in non-survivors were considerably greater at 12, 24 and 48 hours following surgery, according to studies on

hemodynamically stable surgical patients. Research by Nguyen Hb et al., The sensitivity and specificity for predicting in-hospital mortality were attained with a lactate clearance cutoff of less than 10%..[8]

The lactate clearance was computed by dividing the lactate at ED presentation by the lactate at the ED at one hour, subtracting the lactate at six hours, and then multiplying the result by 100Individuals were allocated to either the low clearance group (less than 10% lactate clearance) or the high clearance group (more than 10% lactate clearance). [9]

The goal of this study was to determine the predictive usefulness of plasma lactate levels and lactate clearance in children with severe sepsis.

Aims and Objectives

Aim of the Study

To determine the blood lactate levels in patients with suspected sepsis admitted to pediatric intensive care unit of SHRI BM PATIL MEDICAL COLLEGE.

Objective of the study

Primary objective

To determine the blood lactate levels in patients with sepsis admitted to a paediatric intensive care unit and to correlate with their hospital outcomes.

Secondary objectives

1)To analyse the correlation between initial serum lactate levels and the sepsis severity(e.g.: sepsis, severe sepsis, septic shock) .

2)To determine serial serum lactate levels over time and its association with patient outcomes.

3)To assess the utility of lactate clearance (percentage reduction in lactate levels over a defined period) as a predictor of recovery or deterioration.

Review of literature

"Septicaemia is a state of microbial invasion from a portal of entry into the blood stream which causes sign of illness," Schott Mueller noted in 1914. Since sepsis and septicaemia were used to describe a number of vague clinical symptoms that were present in bacteraemia patients, the definition remained relatively constant over time. Less than 50% of patients exhibiting sepsis symptoms in clinical settings obtain positive blood culture results. Furthermore, sepsis and septicaemia are not the same thing because not all individuals with bacteraemia exhibit sepsis symptoms.

Multiple organ dysfunction syndrome (MODS) and death can result from sepsis, which ranges in severity from infection and bacteraemia to sepsis and septic shock. At the other end of the spectrum are multiple organ dysfunctions, which are progressive, process-based physiological abnormalities

in individual organs. Organ function can change in a variety of ways, ranging from minor malfunction to complete organ failure.

Although sepsis is one of the leading causes of death globally, accurate population-level data collection is difficult. When the body's immune system reacts excessively to an infection, it can lead to organ malfunction and sepsis, a potentially fatal illness. The body's immune response harms its own organs and tissue and if it is not identified and treated right once, it may result in shock, multiple organ failure, and even death. Although everyone can have sepsis, those who are elderly, very young, pregnant, or have other health issues are more vulnerable.[10.]

Originally, the Systemic inflammatory response syndrome (SIRS) scale was used to evaluate sepsis. However, as SIRS can occasionally be a host adaptive response and nonspecific, SIRS-based observation and sepsis detection may not be comprehensive. Therefore, in order to define sepsis, other criteria that are not based on SIRS have also been taken into account.

Pyrexia and neutrophilia are examples of nonspecific SIRS criteria that are useful in the diagnosis of infection. The complicated condition known as sepsis occurs when immune cells destroy one's own tissue. This frequently manifests as organ malfunction and failure. When treating sepsis, it's also important to take into account the different cellular abnormalities that underlie each organ failure. Therefore, fresh definitions of sepsis have been proposed based on these criteria.

The condition known as sepsis occurs when severe organ failure happens because the host immune response fails to regulate during an infection. The immune system of humans along with infectious bacteria act as agents that cause sepsis illness development. The Sepsis Related Organ Failure Assessment (SOFA) score functions as the main tool for examining organ dysfunction. The risk of death increases when patients have higher values on the SOFA score. The SOFA score operates

between 0 and 4 while reaching its highest possible outcome at 24 points representing six-organ dysfunction categories. Measurement of systolic blood pressure below 100 mmHg together with altered mental state and persistent respiratory rate above 22/min constitutes the QSOFA criteria for sepsis assessment.[11]

Epidemiology

Between 1 to 26% of children admitted to hospitals have severe sepsis and septic shock. Mortality is high, with rates as high as 35% in underdeveloped nations and 5% in developed nations. The World Health Organisation Report, one of the most important sources of data for global health policy decision-making., does not include sepsis as a cause of death, and it lacks an adequate disease definition, which are major barriers to a better understanding of sepsis epidemiology in children. [12]

The World Health Organization designated sepsis, which is the body's potentially fatal reaction to infection, as a worldwide health concern in 2017. According to a new analysis by the Global Burden of Disease group, there were twice as many deaths and 49 million instances of sepsis in 2017 as previously thought. Both sepsis incidence alongside its mortality statistics come mostly from high-income countries since low- and middle-income countries lack sufficient data for analysis. The improper assessment of sepsis mortality becomes problematic since LMICs hold 87% of Earth's total population. The study of sepsis epidemiology remains limited in India because of its 1.34 billion population despite its high rates of disability and death. The problem in India intensifies because of an extreme level of antimicrobial resistance which functions as an important health problem throughout the nation.. [13]

Systemic inflammatory response syndrome (SIRS)[14,15]

Must have at least 2 of the following of which at least one must be abnormal temperature or abnormal leukocyte count:

1. Abnormal heart rate (HR) defined as tachycardia or bradycardia .
2. Tachypnea >2 SD above normal for age, mechanical ventilation for processes other than anaesthesia, or underlying neuromuscular illness
3. Abnormal temperature is characterised as fever (core temperature >38.5°C) or hypothermia (core temperature <36°C).
4. Abnormal leukocyte profile, such as increased or depressed numbers for age (not from chemotherapy), or >10% immature neutrophils.

Infection

A suspected infection or one confirmed by positive culture, tissue stain, or molecular testing produced by any pathogen, or a clinical condition associated with a high risk of infection. Acceptable evidence can include physical exam, laboratory, or radiologic findings. Sepsis (SIRS) is a condition caused by infection.

Severe sepsis

Sepsis accompanied by cardiovascular organ dysfunction, acute respiratory distress syndrome (ARDS), or two or more additional organ dysfunctions.

Septic shock

When sepsis manifests according to the above definition such conditions along with

cardiovascular organ failure signs should persist following initial fluid therapy (40 ml/kg intravascularly given within one hour). OR

At least two of the following

- Unexplained metabolic acidosis: base deficit >5.0 mEq/L;
- Increased arterial lactate >2 times upper limit of normal
- Oliguria: urine output <0.5 mL/kg/h
- Prolonged capillary refill: >5 s
- Core to peripheral temperature gap >3 °C

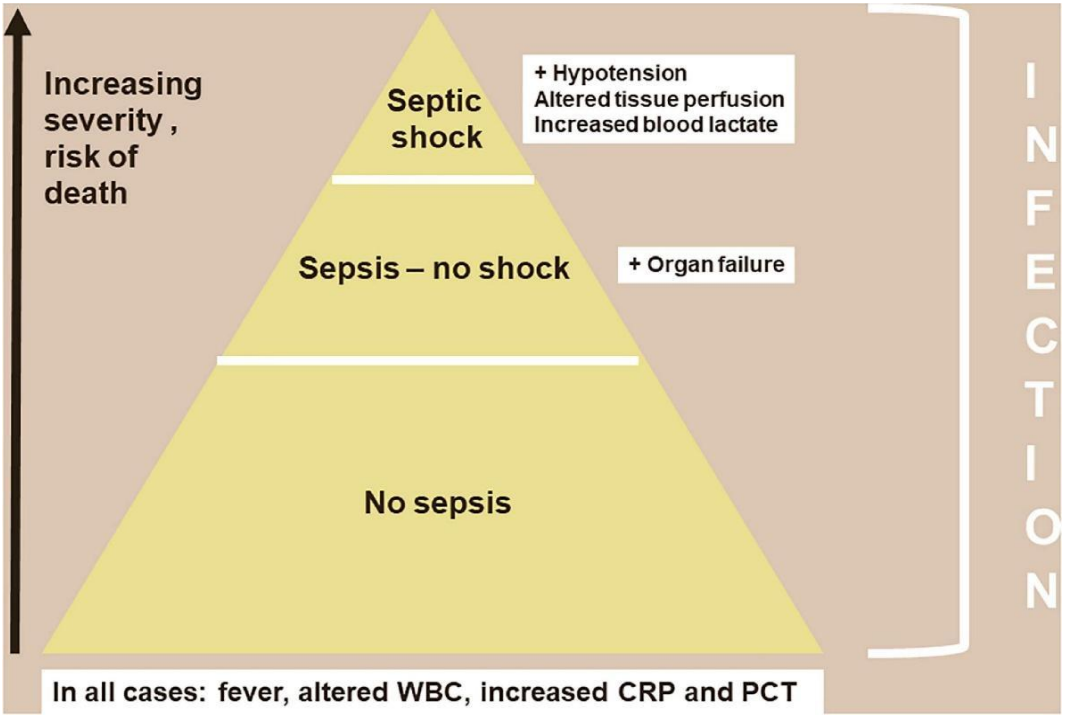

Figure 1: Systemic inflammatory response syndrome (SIRS)

Pathophysiology [15]

The clinical state of sepsis exists at several stages between the systemic inflammatory response syndrome (SIRS) and the multiorgan dysfunction syndrome (MODS) that develops before death.

Several markers signify that inflammation is initiating.

Subjective and objective clinical findings include hypothermia beneath 36 degrees Celsius while also experiencing fever above 38 degrees Celsius. Additionally tachycardia manifests as a heart rate exceeding 90 beats per minute together with tachypnea when breathing occurs more than 20 times per minute and leukopenia presents as WBC levels below 4000 per cubic millimeter or leucocytosis if WBC levels exceed 12000 per cubic millimeter.

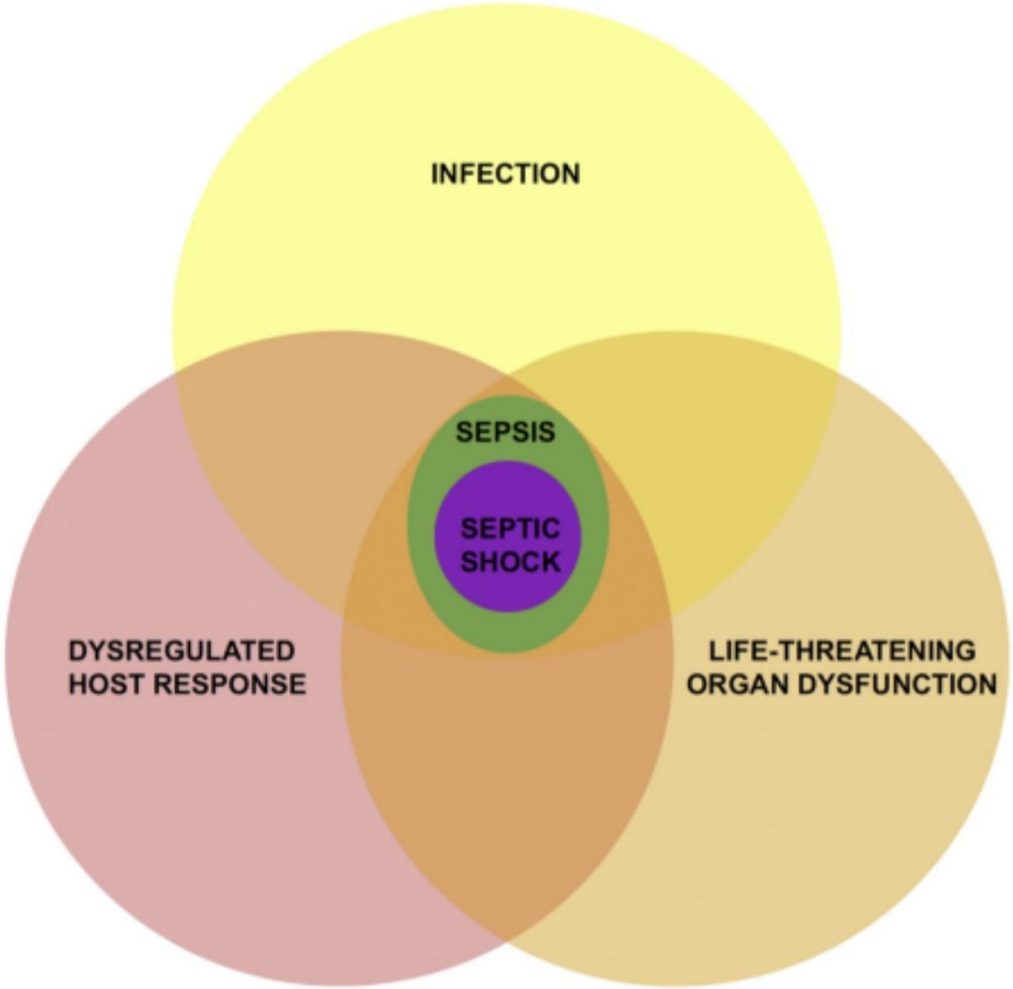

Figure 2 : Sepsis

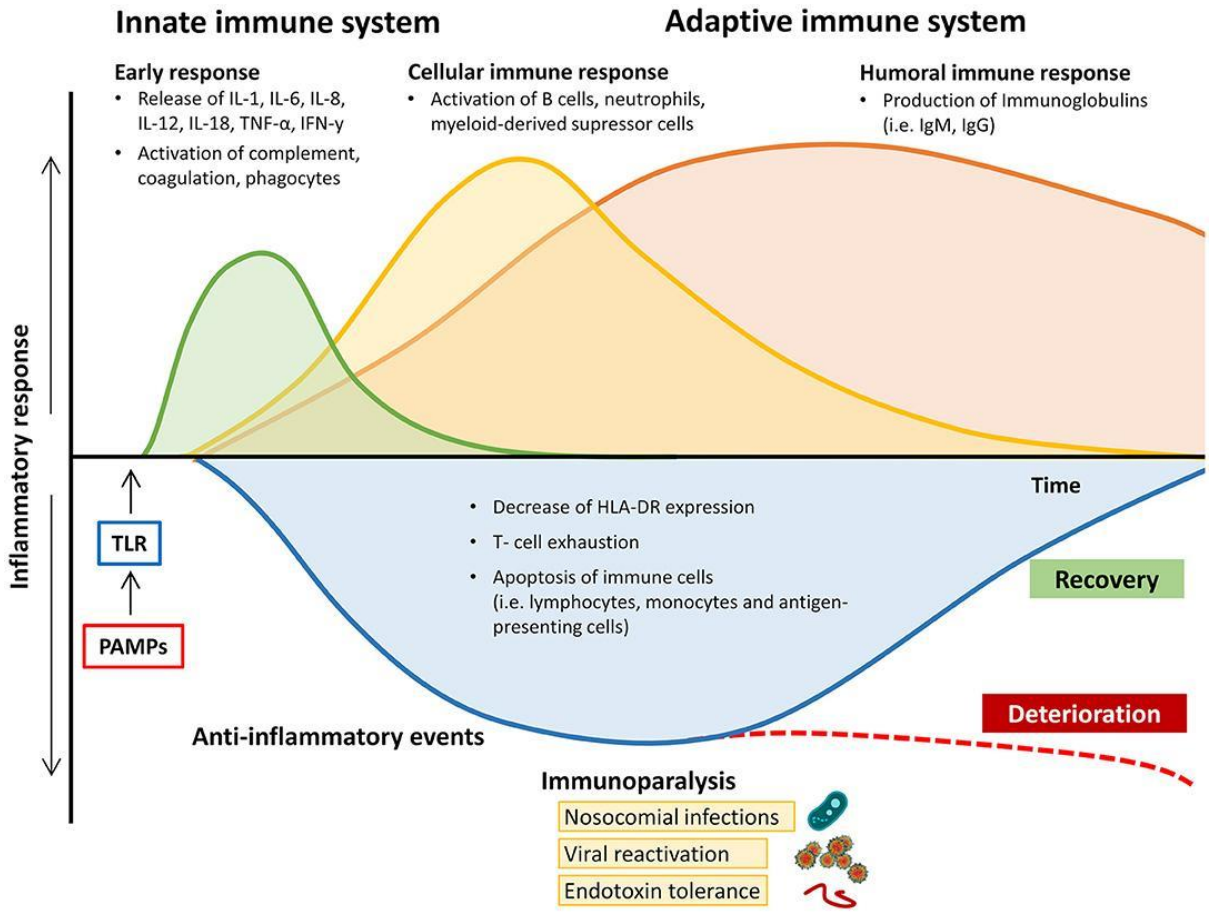

Figure 3 : Immune response to sepsis

Hypovolemia is brought on by capillary leakage, vasodilation, and fluid loss to the third space in sepsis. The signs of insufficient oxygen delivery together with tissue perfusion failure appear often in septic children across their skin and brain along with their kidney tissues. The first signs of sepsis are often obscure which makes it very difficult to detect the illness in children. Tachycardia emerges as a frequent nonspecific warning sign that medical staff often detect during the first stages of shock. Infants along with children show hypotension as a sign of shock only in advanced stages thus its absence during diagnosis remains acceptable. Cardiac output stability in children is

primarily dependent on heart rate elevation and venous tone increase and systemic vascular resistance elevation although they show minimal capacity to boost myocardial stroke volume..[15]

A child must be septic if they have a proven or suspected illness and exhibit symptoms of a systemic reaction to it. Diagnoses of end organ system involvement are necessary for severe sepsis. Cardiovascular dysfunction must be present in septic shock and cannot be addressed by first fluid resuscitation. These definitions sought to prevent a severe, perhaps fatal inflammatory response to infection and to stop the spread of infection by detecting sepsis early enough to enable early management. [16.]

The definition of sepsis in children and adults differs significantly in that the laboratory markers related to physiologic and organ systems have age-specific cutoffs. Extreme tachycardia can be used for an extended period of time by the healthy kid cardiovascular system to sustain cardiac output without causing myocardial ischemia. In children, hypotension typically manifests later than in adults and frequently signals an impending, possibly irreversible circulatory collapse. In order to prevent cases of severe decompensation that could ultimately end in mortality, the pediatric consensus guidelines are made to identify kids who are experiencing compensated septic shock.

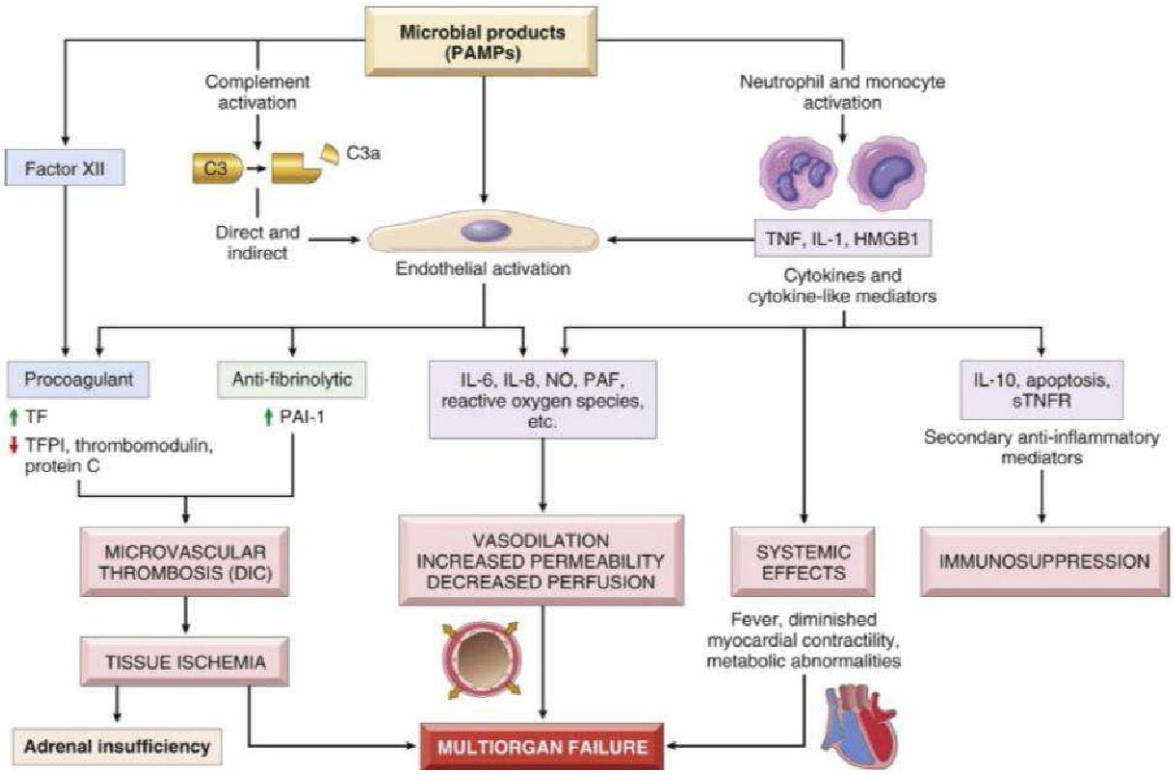

Figure 4: Showing Multiorgan failure due to sepsis

Response to Infection by the Developing Immune System.[16]

Complete immunologic maturation occurs during adolescence when the immune systems of children become substantially different from adult systems. Immune system function requires substantial evolution from the sterile intrauterine environment to encounter the complex and changing microbial environment which will shape the rest of life for the newborn. The immune response of neonates remains weak because their systems present the most severe impairment. Neonatal phagocytes exhibit weakened activity for cell adhesion and extravasation besides producing lower amounts of inflammatory cytokines while presenting fewer antigens to adaptive immune cells and displaying less PAMPs sensitivity when compared to adult phagocytes. The

activity of NK cells is reduced in addition to their poor cytotoxic abilities and the quantity of complement is limited to 10–70% compared to adult amounts..[17]

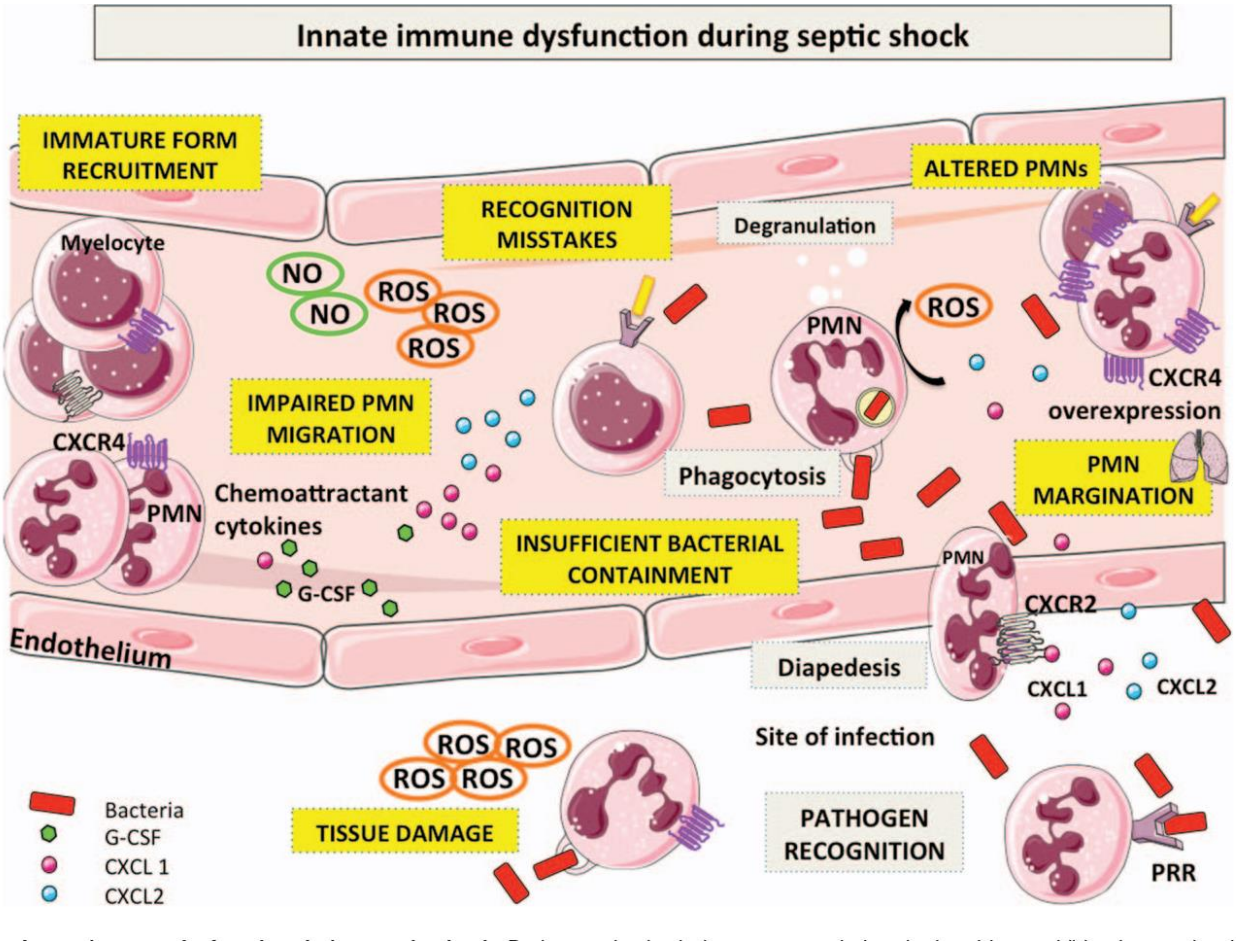

Figure 5: Innate immune response during septic shock

Additionally, the very young have decreased adaptive immunity. Neonates have significantly larger T cell counts than adults, but their functioning is often subpar, partly because they produce less interleukin-2 (IL-2). Because the neonate produces less interferon-γ (IFNγ), cytotoxic CD8+ T cells are less active and helper CD4+ T cells are toward Th-2 (humoral) responses. Although they

are also common in neonates, B lymphocytes are primarily naïve, primarily generate IgM immunoglobulins and have a low sensitivity to capsular polysaccharides. Though full immune competence is not attained until adolescence, adaptive and immune responses have substantially approached those of healthy adults by the time a child is two years old.

Infants and young toddlers are significantly more vulnerable to serious infections from a variety of species, especially viruses and encapsulated bacteria, as a result of these immune function deficiencies. Children under two years old are particularly vulnerable to severe viral infections, partly because of unregulated viral replication brought on by decreased cytotoxic lymphocyte responses and IFNγ production. Trans placentally acquired maternal antibodies, mainly IgG and IgA, are the main mitigating factor during the first six months of life. For this reason, the recommendations for maternal immunization should be expanded to address common infections linked to severe paediatric illness.

Furthermore, the use of protein-polysaccharide conjugate vaccines can encourage the production of protective levels of immunoglobulin by newborn immune systems against pathogens encapsulated in polysaccharides, such as Streptococcus pneumoniae (S. pneumoniae) and Haemophilus influenzae type b (Hib).[18] The community can benefit from such vaccines since they can even alter the way pathogens are carried in the nasopharynx. Since toddlers under the age of two are not as immunogenic as older children and adults, vaccinations against common viral pathogens especially influenza are regrettably not licensed for use in children under the age of six months. [19].

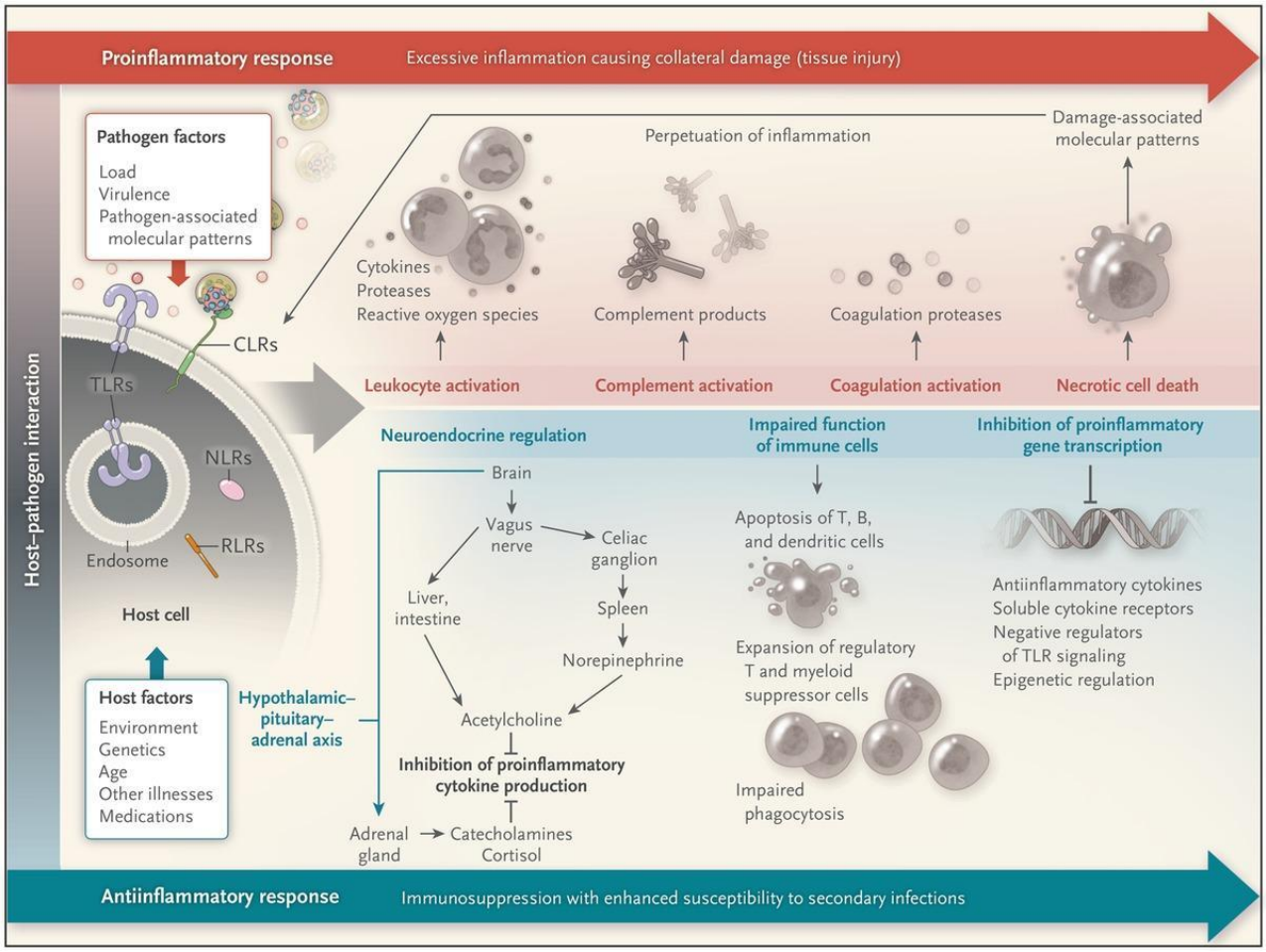

Figure 6: Host Pathogen interaction

Epidemiology and Clinical Manifestations of Pediatric Sepsis

The most prevalent bacterial pathogens in newborns with late-onset sepsis are enteric gram-negative rods, particularly Escherichia coli, and group B streptococci (GBS). Protocols for peripartum prophylaxis have decreased the prevalence of sepsis associated with GBS. In young infants, Bordatella pertussis can induce a serious sickness that is marked by frequent episodes of gagging, apnoea, cyanosis, and bradycardia. The condition leads to death at high rates when

pulmonary hypertension and respiratory failure develop. Mass distribution of conjugate vaccines prevents H. influenzae type B from persisting as one of the primary bacterial sepsis causes in young children who make up five percent of the population worldwide. S. pneumoniae stands as the leading cause of pneumonia hospitalization but the conjugate vaccination protects children against 7-valent and 13-valent S. pneumoniae strains that cause childhood pneumonia.

vaccination against pneumoniae decreases the likelihood of invasive bacterial infections to a level of 76% when used. Methicillin-resistant (MRSA) strains in communities reduce the effectiveness of standard antibiotics and treatment times which makes S. aureus one of the primary pathogens that leads to invasive illness requiring hospitalization of children. The death risks increase for sick children but antimicrobial resistance in gram-negative enteric bacteria and opportunistic pathogen groups (Pseudomonas, Acinetobacter, and Burkholderia spp.) causes medicine delays and rises virulence levels in certain multidrug-resistant strains.

The natural course of multiple viruses depends on patient age together with their immune system health to cause viral-induced sepsis. Drinking the flu virus stands as one of the most prevalent triggers for juvenile viral sepsis while accounting for the worst cases of hospital admissions and deaths. Even with vaccination success rates preventing most influenza-induced serious respiratory infections, inadequate vaccination levels and vaccine-response deterioration among young children along with vaccine-strain mismatch result in long-term healthcare system stress. Parainfluenza virus leads to severe pneumonia in infants along with children who have weak immune systems and respiratory problems but mainly affects the upper airway of healthy children as it causes croup.

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Risk for sepsis

- Being a newborn baby, mainly with low birth weight or prematurity
- Cancer- leukaemia, or chemotherapy
- Diabetes or a neuromuscular disorder
- A problem with the immune system

Sepsis's early symptoms, which might include fever, cough, sore throat, vomiting, and diarrhoea, are frequently non-specific. Any combination of the following symptoms could appear as the condition worsens:

- Shivering, fever, or extreme coldness
- Severe discomfort or pain
- Hot or clammy skin
- Disorientation or confusion
- Dyspnoea
- Elevated heart rate

Sepsis's early symptoms frequently mimic those of a viral disease. Because of this, diagnosing sepsis is challenging (Journal of Family Health Care, December 2016).

However, parents, caregivers, and medical professionals need to act quickly if a kid exhibits any of the following **"red flag"** symptoms:

- Pale
- Blueish, or mottled skin

- Sluggish or difficult to wake up
- Unusually cold
- Breathes rapidly
- Rash that doesn't go away when you touch it.
- Seizure or fits

Children and infants are frequently difficult to evaluate since they will physically compensate for a long time until they become overwhelmed and quickly deteriorate. Children under one year old are more likely to develop sepsis, including:

- Preterm infants (less than 37 weeks)
- Newborns whose membranes ruptured for an extended period of time (greater than 24 hours before to delivery)
- Babies whose moms had a fever (over 38°C) during labour
- Children who have had invasive procedures, trauma, or injuries within the last six week.
- Children whose immune has been compromised by disease or medication (such as steroids or chemotherapy)
- Children who have indwelling lines or catheters or who have any skin integrity violations (such as burns, scrapes, blisters, or infections)
- youngsters who have not received all of their recommended vaccinations in accordance with the national schedule NICE Guideline [NG51]: Sepsis: detection, diagnosis and early

care.[21] for more specific information on sepsis in children, including symptoms and signs.

Lactate

Carl Wilhelm Scheele in 1780 discovered lactate which has frequently been misidentified as a simple hypoxic waste product that results from oxygen deprivation during muscle contractions, which can have numerous negative consequences. Based on then-groundbreaking research on frogs by Nobel Prize laureates and on humans, the "low O2 induces high lactate" paradigm has led to the oversimplified conclusion that "high lactate means low O2." Glycolysis is a basic metabolic activity that takes place in the cytoplasm of cells and produces the molecule lactate. The first step in this process is the glycolysis of circulatory glucose, which results in the formation of two pyruvate molecules. [22]

During the enzymatic reaction ATP functions as the primary cellular energy source while generating small yet vital amounts that enable cellular processes. The body shows elevated ratios between pyruvate molecules and lactate molecules during vigorous exercise and stressful circumstances compared to rest periods. In order to regenerate nicotinamide adenine dinucleotide (NAD+), lactate dehydrogenase (LDH) enzymes are essential. Their activity facilitates this conversion process. The maintenance of glycolysis and the satisfaction of the cell's energy needs depend on this NAD+ regeneration. It is significant to note that lactate is effectively delivered into the bloodstream and does not remain there. The main type of lactate in this extracellular environment is lactate anions. This phenomenon, a critical component of the body's metabolic regulation, allows for the efficient exchange of lactate between various tissues and organs, ensuring that energy needs are supplied even in the face of adversity.[22]. The enzyme LDH produces and

breaks down lactate into pyruvate from both L- and D-stereoisomers. But because LDH is stereoselective, L-LDH is necessary for L-lactate to be produced and metabolized, while D-LDH is necessary for D-lactate to be produced. Lactate's normal blood concentration ranges between 1 and 2 mM12, with L-lactate being the predominant enantiomer. The human body's lactate level at rest is about 1 mm. Under normal conditions, the plasma ratio of D-lactate to L-lactate is approximately 1:100. Unless[22,23]

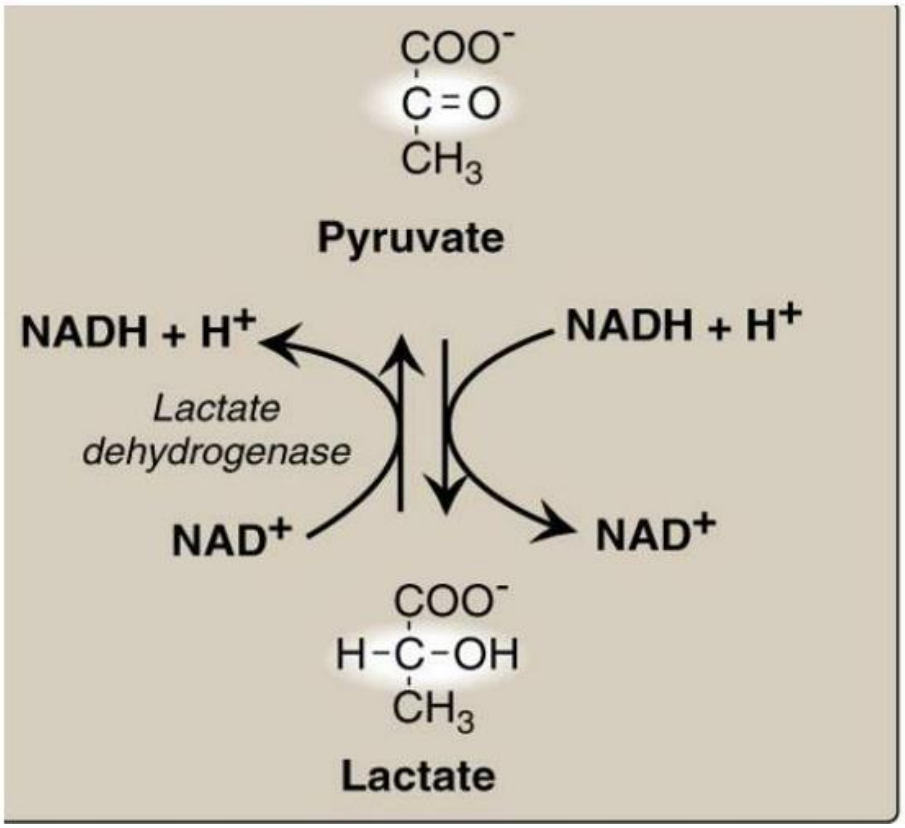

Figure 7: Interconversion of lactate and pyruvate

Lactate shuttle theory[24]

The idea of lactate shuttles explains how it contributes to cell signalling and transports oxidative and gluconeogenic substrates. According to the lactate shuttle idea, this relationship crosses cells, tissues, and organs, breaking down compartmental barriers. In a complex feedback loop, lactate is

viewed as a messenger that increases lactate synthesis in response to transient disturbances in ATP supply. This phenomenon is critical for short- and long-term cellular adaptations to maintain ATP homeostasis. These include the "intracellular lactate shuttle" as well as "cell-cell lactate shuttle"

The majority, if not all, of intracellular and cell-to-cell lactate shuttles are triggered by a redox status, concentration, or pH gradient. Monocarboxylate transporter (MCT), a bidirectional asterospecific pH-sensitive transport protein, can transport lactate across the cell membrane by free diffusion of undissociated acid. Depending on concentration and pH variations, tissues can alternate between releasing and absorbing lactate. Energy supply, glycemia maintenance, cerebral metabolism, and signaling are all impacted by lactate metabolism, which is ubiquitous and has consequences for a number of clinical disorders, including inflammation, heart failure, traumatic brain injury (TBI), and glucose regulation [25]

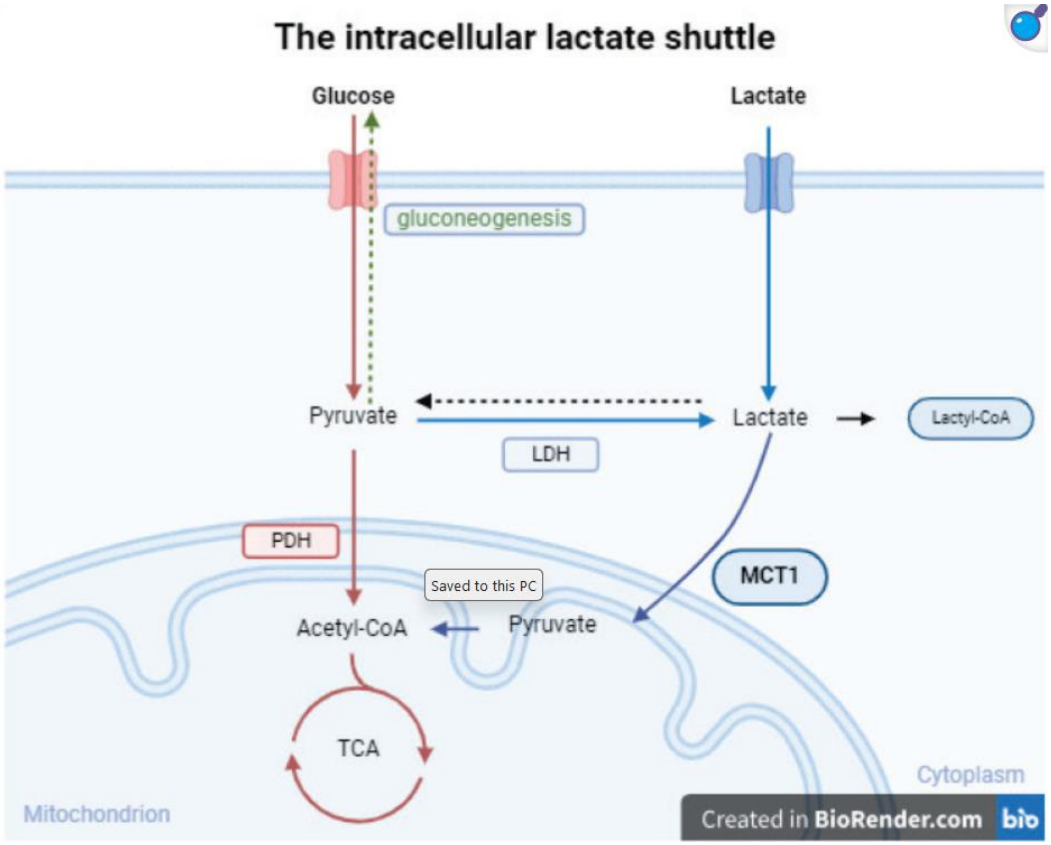

Figure 8 : Lactate shuttle theory

Physiology and Pathophysiology

The majority of bodily tissues create lactate, with muscle producing the most of this substance. Under typical circumstances, the liver quickly eliminates lactate, with the kidneys contributing a tiny amount of extra clearance. Under aerobic conditions, lactate is mostly avoided by producing pyruvate through glycolysis before it enters the Krebs cycle. Lactate is a byproduct of glycolysis that enters the Cori cycle as a substrate for gluconeogenesis when anaerobic conditions are present . L-lactate and D-lactate are the two isomers of lactate. When bacteria in the human colon are exposed to high levels of unabsorbed carbohydrates, they create D-lactate. An excess of D-lactate is produced when there is a change in the intestinal flora and a high carbohydrate load (as in short

bowel syndrome). This can enter the bloodstream and perhaps result in neurologic symptoms. Since D-lactate's function has already been discussed elsewhere, it will not be covered in this review.

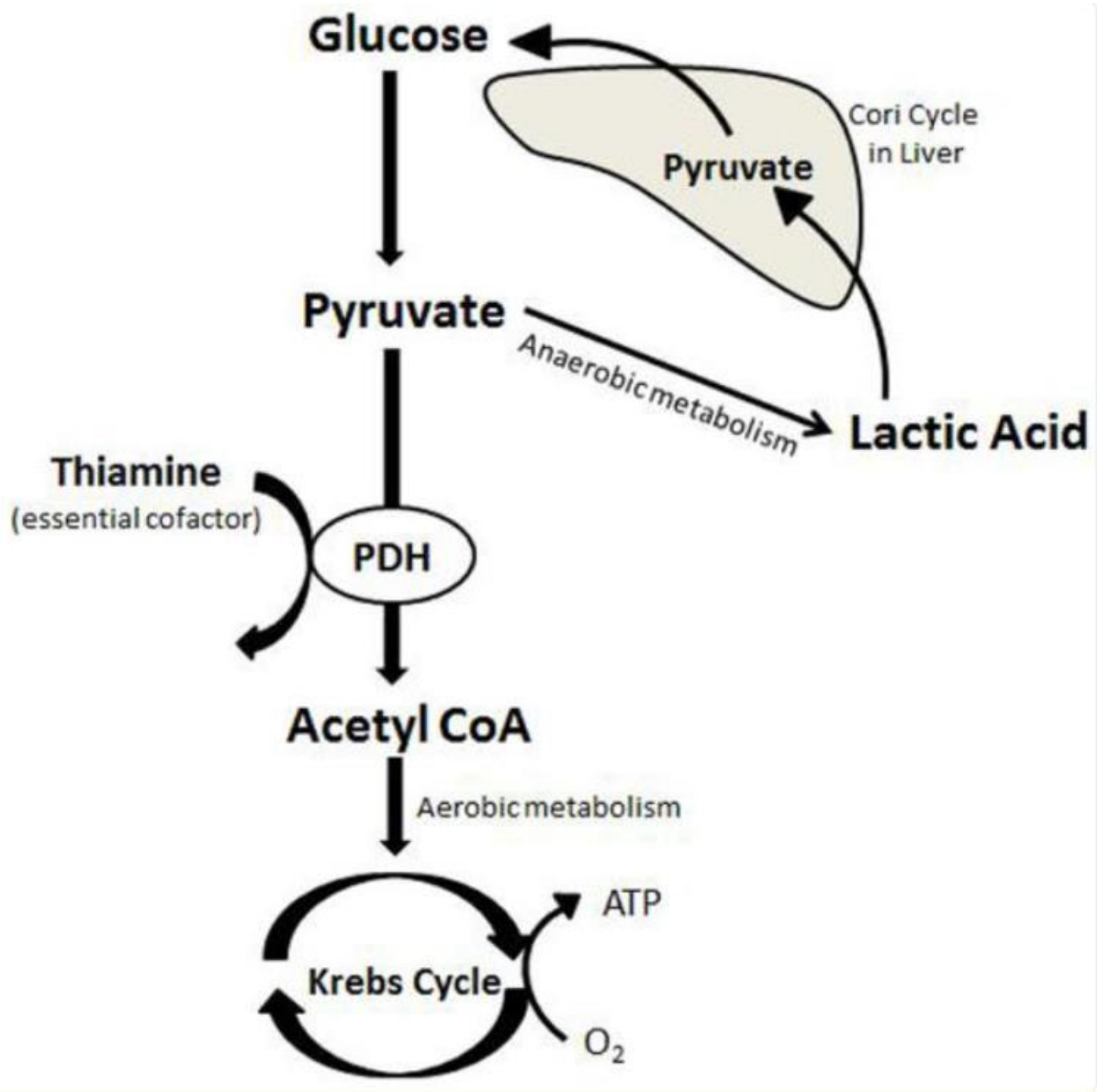

Figure 9: Krebs cycle

Several research studies define "high" lactate as any measure above 4 mmol/L but the definitions for elevated lactate remain inconsistent between 2.0 and 2.5 mmol/L7.8–11 In addition the "normal" value may vary depending on the method of testing. Lactic acid represents the formal acid substance whereas blood tests detect lactate as a basic compound despite common interchange between these terms. Although "lactic acidosis" is frequently used in clinical settings to refer to high lactate, it should only be used in situations when a matching acidosis (pH < 7.35) is present. Elevated lactate in different situations likely has a complex, patient-specific, and disease-specific etiologic. Elevated lactate might result from either slower clearance, higher generation, or a mix of the two. In shock conditions, the cause of high lactate is arguably well studied. Several mechanisms seem to be involved, such as mitochondrial malfunction (including the possible absence of essential enzymatic co-factors), the presence of a hypermetabolic state, and hypoperfusion brought on by macro- and/or microcirculatory dysfunction. Increased production and reduced clearance are two effects of liver dysfunction that become even more significant under hypoperfusion conditions.

Lactate in sepsis[27,28]

In critically ill individuals, lactate is a non-specific indicator of the severity of the illness. One Blood lactate levels are typically low, and high levels can signify either a maladaptive or protective reaction to shock.[27] Patients who are in the early stages of deterioration, with reduced organ perfusion, but who are maintaining their blood pressure and whose vital signs and behaviour may otherwise be misleadingly comforting, can be identified with the help of elevated lactate levels and failure to remove lactate.[28] In deteriorating adult patients, elevated lactate levels have been demonstrated to predict outcomes after sepsis. One Other forms of shock, such as haemorrhagic or

cardiogenic shock, or other catecholamine reactions, such seizures and hyperthermia, can also have an impact on lactate. Drugs like metformin, salbutamol, adrenaline infusions, and antiretroviral (HIV) therapies can also increase lactate.

Thresholds for lactate levels

All age groups typically have lactate levels below 1.0 mmol/L.

Over 2.0 mmol/L of lactate indicates high lactate levels, which should be evaluated by a physician skilled in treating patients who are deteriorating and treated as prescribed.

A lactate level of 4.0 mmol/L necessitates immediate treatment and escalation via the fast response system of the health service organization, as it considerably raises the risk for both morbidity and mortality.

A lactate level below 2 mmol/L does not rule out a sepsis diagnosis, particularly in a pediatric patient, at low systolic blood pressure, or in the presence of organ dysfunction symptoms.

Causes of increased lactate level

For many years, prognostic scoring systems for septic patients have included SOFA and APACHE II scores. However, a lot of research was done to find the most straightforward and effective prognostic marker because of laboratory limitations. As a result, serum lactate measures have become increasingly important in recent years. Even while lactate monitoring is emphasized, there

is currently little proof that both initial and serial lactate monitoring can produce improved outcomes[29].

Lactate levels may rise as a result of several circumstances that cause insufficient oxygen delivery, exaggerated oxygen demand, and reduced oxygen usage. A hallmark of sepsis and septic shock is hyperlactatemia. It is claimed that the mechanisms underlying hyperlactatemia in the two disorders differ. In sepsis, an elevated lactate level indicates a higher glycolytic flux brought on by hypermetabolism; in septic shock, tissue hypoxia causes an enhanced glycolytic flux.[30] According to this, there are two types of lactate: "stress lactate" and "shock lactate."[31,32] Critically ill patients typically have blood lactate levels up to 18 mg/dl (2 mmol/l) as normal.

Lactic acidosis is defined as lactate levels above 45 mg/dl and pH below 7.35, while hyperlactatemia is characterized as lactate levels between 18 and 45 mg/dl without metabolic acidosis.[32] A potentially reversible state, such as early septic shock, may be identified by elevated lactate levels, which could suggest that "there is still room" to improve prompt action.[32] One significant and extensively researched predictive indicator of death in individuals with sepsis or septic shock is the lactate level. Studies examining lactate levels in children with sepsis or septic shock are scarce. Blood lactate concentration in neonates requires independent measures because research has indicated a weak association between it and pH or base excess.

Although hyperlactatemia in preterm infants has been identified as a sign of sepsis, it is unclear how predictive this condition is of the prognosis. Lactate's effects in pediatric sepsis/septic shock are not entirely consistent. Hatherill et al.33 observed no noticeable difference in lactate levels between survivors and non-survivors, but Duke et al.34 discovered lactate to be a good predictor of mortality. The current study was conducted to investigate serial lactate levels in children suffering from septic shock and correlate these levels with the outcome. A variety of factors might contribute

to increased lactate levels. Recently, the majority of medical work on the significance of lactate levels has concentrated on septic shock.

Septic shock

In addition to microcirculatory failure and reduced oxygen and nutrient extraction by peripheral tissues, septic shock is frequently linked to macro circulatory dysfunction that results in arterial hypotension. Levels of lactic acid have emerged as a valuable indicator of tissue hypoperfusion and could be used as a resuscitation endpoint in sepsis and septic shock patients. Shapiro et al[35]**.** discovered that lactate levels may accurately stratify patients based on mortality in a research involving 1278 patients who were admitted with an illness. Mortalities of 4.9% (95% CI: 3.5% – 6.3%), 9.0% (95% CI: 5.6% – 12.4%), and 28.4% (95% CI: 21% – 36%) were linked to lactate levels of 0–2.4, 2.5–3.9, and ≥4 mmol/L, respectively.

Cardiogenic, Obstructive and Haemorrhagic Shock

Although myocardial dysfunction leading to shock following heart surgery revealed significantly higher lactate levels, the usefulness of lactate in cardiogenic shock has not been thoroughly examined. Researchers discovered that rather than decreased clearance, the spike was mainly caused by increased tissue lactate synthesis. Lactate has been identified as a valuable determinant

of mortality in individuals with cardiogenic shock who need extracorporeal membrane oxygenation.[35] patients with inadequate lactate clearance (<10%) in cardiogenic shock after ST-elevation myocardial infarction the survival rate will be reduced . A pulmonary embolism may also be associated with elevated lactate. Vanni et al[36]. demonstrated that, regardless of hemodynamic condition or right ventricular dysfunction, high lactate (>2 mmol/L) was linked to increased mortality.

Haemorrhagic shock

Elevated lactate can also be caused by haemorrhagic shock. When lactate levels were assessed in 60 patients who arrived at the emergency room, Akkose et al[37]. discovered that both traumatic and non-traumatic haemorrhagic shock patients had considerably higher lactate levels than controls, with the traumatic group who are having the highest value. The study lacked sufficient power to identify any variation in mortality.[37]

Cardiac Arrest[38]

Lactate's function in the population during cardiac arrest has also been investigated. The first increase in lactate is most likely caused by both ischemia from a lack of blood flow during arrest and inflammation from ischemia-reperfusion injury. Microcirculatory dysfunction, myocardial stunning leading to cardiogenic shock, systemic inflammatory response and persistent tissue hypoxia, an uncorrected underlying aetiology of the initial arrest, and mitochondrial injury and dysfunction are some of the causes of persistently elevated lactate in the post-arrest period. In a post-arrest

retrospective cohort, the initial lactate level and the requirement for vasopressor treatment within the first few hours after the arrest could be used to stratify patients and predict outcomes with high accuracy.

In a post-arrest retrospective cohort, the initial lactate level and the requirement for vasopressor treatment within the first few hours after the arrest could be used to stratify patients and predict outcomes with high accuracy. Death rates for post-arrest individuals with an initial lactate of less than 5 mmol/L were 39%, whereas those with an initial lactate of more than 10 mmol/L had a 92% death rate. Moreover, two investigations of post-arrest patients found that the capacity to eliminate lactate during the post-arrest phase was a predictor of improved survival.[38]

Trauma

Patients who have had traumatic injuries frequently experience hypoperfusion, which is most frequently caused by blood loss. The absence of vital sign abnormalities does not rule out occult hypoperfusion, even though their presence may aid in the diagnosis of shock. Those who initially show normal vital signs may conceal persistent tissue hypoperfusion may be identified with the aid of lactate rise.

Lactate levels has been observed to be considerably greater in non-survivors of trauma than in survivors, similar to sepsis and cardiac arrest. According to one study, patients with thoracic trauma and a lactate level more than 4 mmol/L have an estimated sensitivity of 84% and specificity of 86% for mortality. Following traumatic injury, the risk of multi-organ dysfunction and survival is highly correlated with the level of increased lactate and the pace of lactate clearance; lactate clearance may also provide end point to guide resuscitation.

Seizure

Depending on the kind of seizure, lactate levels can rise significantly. The clinician should be aware that elevated lactate levels in this situation are temporary. Lactate production stops and is quickly eliminated when the seizure has passed. More research is necessary if the lactate level remains elevated for longer than the typical 1–2 hours after a seizure. This could indicate a separate or concurrent underlying cause.

Excessive Muscle Activity

Because of anaerobic metabolism, lactate levels rise during vigorous exercise. Siegel and colleagues found that 95% of collapsed marathon runners had elevated lactate levels ranging from 1.1 to 11.2 mmol/L.Excessive muscle work may contribute to elevated lactate in the context of acute severe asthma. According to Rabbat et al. acute severe asthma frequently results in increased lactate, which rises over the first six hours following admission. They did not discover any correlation with death or the development of respiratory failure. Excessive adrenergic stimulation may also be a factor in the usage of beta agonists to treat asthma. However, more research is necessary to determine the precise pathophysiology of increased lactate in asthma. Additionally, it has been proposed that respiratory muscle fatigue and excessive muscle effort, regardless of the underlying reason, contribute to high lactate; however, additional research is required to elucidate this association.

Burns and Smoke Inhalation

The measurement of lactate proves useful as a strong indicator when evaluating patient prognosis for individuals who suffered serious burns. The initial lactate value proved to be an effective clinical marker that distinguished between burn survivors and non-survivors according to Jeng et al.[40.] The applied cut-off value for initial lactate measurement at 2 mmol/L produced equivalent results in experimental data collected by Kamolz et al.[41]. The removal of lactate from the blood quickly associated with decreased mortality rates according to their findings. Burn patients require measurement of lactate values even though its exact role as a resuscitation end-point remains unclear because sepsis with multisystem organ failure stands as the main contributor to morbidity and mortality in burns treatment. lactate levels affect people who inhale smoke because they could either absorb carbon or cyanide gas.

Thiamine Deficiency[26]

The tricarboxylic acid cycle and aerobic carbohydrate metabolism depend on several biological enzymes, including as pyruvate dehydrogenase and α-ketoglutarate dehydrogenase, which thiamine co-factors. Without thiamine, lactate production rises and anaerobic metabolism takes over. Thiamine insufficiency has been well documented to cause the formation of increased lactate in serum and cerebrospinal fluid.

Anorexia nervosa, liver illness, chronic wasting diseases, alcoholism, hyperemesis gravidarum, and gastric bypass surgery are among the dietary deficiencies that increase the risk of thiamine deficiency. Thiamine deficiency-induced increased lactate is a frequently disregarded but treatable illness that should be taken into consideration in cases of otherwise unexplained elevated lactate.

Liver Dysfunction[26]

The liver is the primary organ in charge of lactate elimination, and it may be compromised in cases of severe liver disease. Furthermore, research has indicated that the liver's acute injury may serve as a source of lactate. Medical personnel must thoroughly investigate and provide proper treatment for alternative treatable causes of elevated lactate levels before diagnosing liver illness from elevated lactate results.

Inborn errors of metabolism seldom cause high lactate levels yet this occurs most frequently in children under medical care. Mutations affecting enzymes that make up the tricarboxylic acid cycle as well as the other metabolic processes like gluconeogenesis and the respiratory chain and pyruvate dehydrogenase normally occur.

Lactate as marker for sepsis[42]

A practical and therapeutically accessible surrogate indicator of tissue hypoxia and disease severity that is not influenced by blood pressure is lactate. Poor vital signs are displayed. A rise in catecholamines and a change in the brain's regulation of arterial pressure maintenance are caused by decreased tissue perfusion, which also affects resuscitation endpoints and outcome prognosticators . Lactate levels are one of the key indications included in the surviving sepsis campaign (SSC) guidelines. This main set of parameters is helpful in forecasting the mortality outcome and the degree of sepsis. Lactate clearance is a strong indicator of improvement over mortality, according to SSC guidelines. The degree of clearance within the first six hours is a reliable indicator of the patient's chance of survival. Consequently, lactate clearance was demonstrated to be an independent predictor of the result when the lactate clearance parameter was added to the principal bundle. Several other papers that have demonstrated lactate as a sepsis marker have been compiled.[42]

SL NO	STUDY	POPULATION SIZE	LACTATE (CONTROL)	LACTATE (SURVIOUURS)	LACTATE (DEAD)
	Bakker J et al [43]	87	<2 mmol/L	5.6+3.7mmol/l	9.6+5.3mmol/l
	Howel et al[44]	1287	<2 mmol/L	>4mmol/l	NA
	Lavery et al [45]	375	<2 mmol/L	>2mmol/l	NA
	Nguyen H B el at [46]	111	<2 mmol/L	6.9+4.6mmol/l	NA
	Arnold et el[47]	166	<2 mmol/L	>4mmol/l	NA
	Krishna et al [48]	50	<2 mmol/L	24.4+17.9mg/dl	34.2+19.8MMOL/L

Kana Ram Jat et al discovered that nonsurvivors had greater blood lactate levels at admission, as well as at 12 and 24 hours. A lactate level of greater than 45 mg/dl (5 mmol/l) was a reliable predictor of death. [11]

Another study conducted by **Tejaswini et al** Serum lactate can be employed as an early detection marker in patients with a risk of sepsis, and serial lactate monitoring has the same diagnostic accuracy in predicting outcomes as the established prognostic grading systems SOFA and APACHE II[49].

Materials and methods

Source of data: All cases of suspected sepsis admitted to PICU, Shri BM Patil Medical College Hospital and Research Centre, Vijayapura fulfilling the Inclusion and Exclusion Criteria. Minimum of 100 cases will be studied.

Study design : Case control study

Study period : April 2023 to November 2024 (1.7 years)

Sample size: 100 (50 for each group)

Based on research by Rupak Bhandari, R Bhandari Paudel50, and GB Malla. For sample size computation, use G*Power version 3.1.9.4 programme.

The Lactate (mmol/l) For Sepsis (Mean=2.43, SD=0.893) and dexamethasone (Mean=3.65, SD=1.779), this study requires a total sample size of 100 (50 for each group, assuming equal group sizes), so to achieve a power of 99% for detecting a distinction in Means: Inequality, two

independent means (two groups) (t test) with 5% level of significance.

Selection criteria

Inclusion criteria: All pediatric cases admitted in PICU with suspected sepsis.

Exclusion criteria:

1. Patients who were on antibiotics that caused lactic acidosis were excluded from the study.
2. Patients who are known cases of acidosis.
3. Patients on Anti tubercular drugs.
4. Patients with non-sepsis-related shock, such as cardiogenic, oligemic, anaphylactic, or neurogenic.
5. Patients with known malignancies or on immunosuppressive medications.
6. Patients with severe neurological problems, persistent illnesses, or substantial congenital deformities.
7. Postoperative instances.

Data collection : Children fulfilling the selection criteria were included in the study after obtaining consent from their parents.

Method of study

PATIENT SOURCE

Research took place within a pediatric intensive care unit [PICU] which maintains continuous services for critically sick patients. A minimum group of subjects without sepsis functions as the

control sample. All consecutive subjects joined the study without any exclusions based on age, sex or complaint type.

The clinical history and examinations and investigative reports detailing for every enrolled subject appeared within Annexure 1 of the study. Blood culture and cbc together with random blood sugar level made up the group of investigations used. Medical staff measured arterial blood gas levels for each participant starting from intensive care entrance through 0 hrs , 6 hrs , 12 hrs, 15 hrs and 24 hrs of their admission. The enzymatic methodology running on the ABG equipment .

STATISTICAL ANALYSIS

The research data was transferred into Microsoft Excel before conducting statistical examinations with the SPSS software Version 20. The results appear in Mean and SD, counts, percentages together with diagrams. For normally distributed

An independent sample t- test will evaluate all normal variables between the groups. The Mann-Whitney U test replaces the regular practice when evaluating variables that fail to follow a normal distribution pattern. The Chi-square test or Fisher's exact test is applied to evaluate Categorical variable differences between two groups. The study will consider results significant when p values become smaller than 0.05. All statistical are performed two-tailed

RESULTS

Table 1: Age wise distribution of study participants

Sl No	Age	Cases n (%)	Control n (%)
1	<12 months	7(13.7%)	13(23.2%)
2	1-5 years	20(39.2%)	22(39.3%)
3	>5 years	24(47.1%)	21(37.5%)
4	Total	51(100%)	56(100.0%)

The age-wise distribution of study participants revealed that the majority (47.1%, n=24) in the case group were above 5 years of age, followed by 39.2% (n=20) in the 1–5 years age group, while the

least proportion (13.7%, n=7) belonged to the <12 months category. In contrast, the control group had a higher representation in the 1–5 years age group (39.3%, n=22). This distribution is illustrated in the accompanying bar diagram.

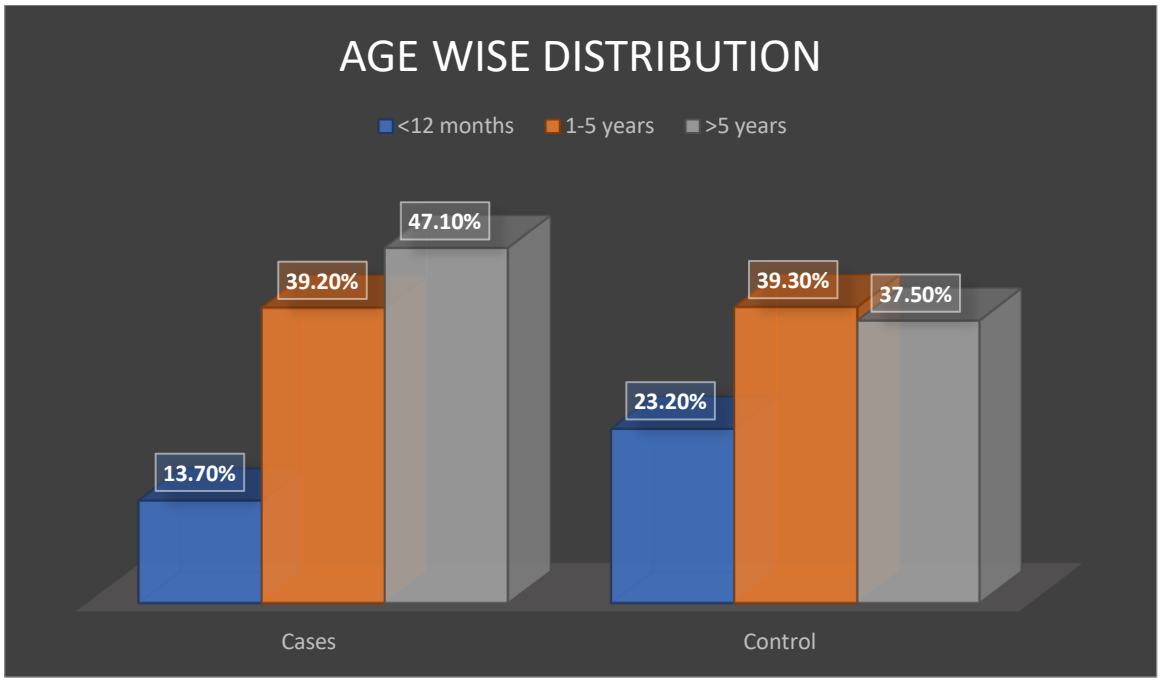

Figure 10: Age wise distribution of study participants

Table 2: Gender wise distribution of study participants

Sl No	Sex	Cases n(%)	Control n(%)
1	Male	28(54.9%)	33(58.9%)
2	Female	23(45%)	23(41.1%)
3	**Total**	**51(100%)**	**56(100.0%)**

The table presents the gender distribution among study participants. In the case group, the majority were males, accounting for 54.9% (n=28) of the 51 children. Similarly, in the control group, 58.9% were males. The gender distribution is fairly balanced between the case and control groups. A pie chart illustrates the gender distribution among cases.

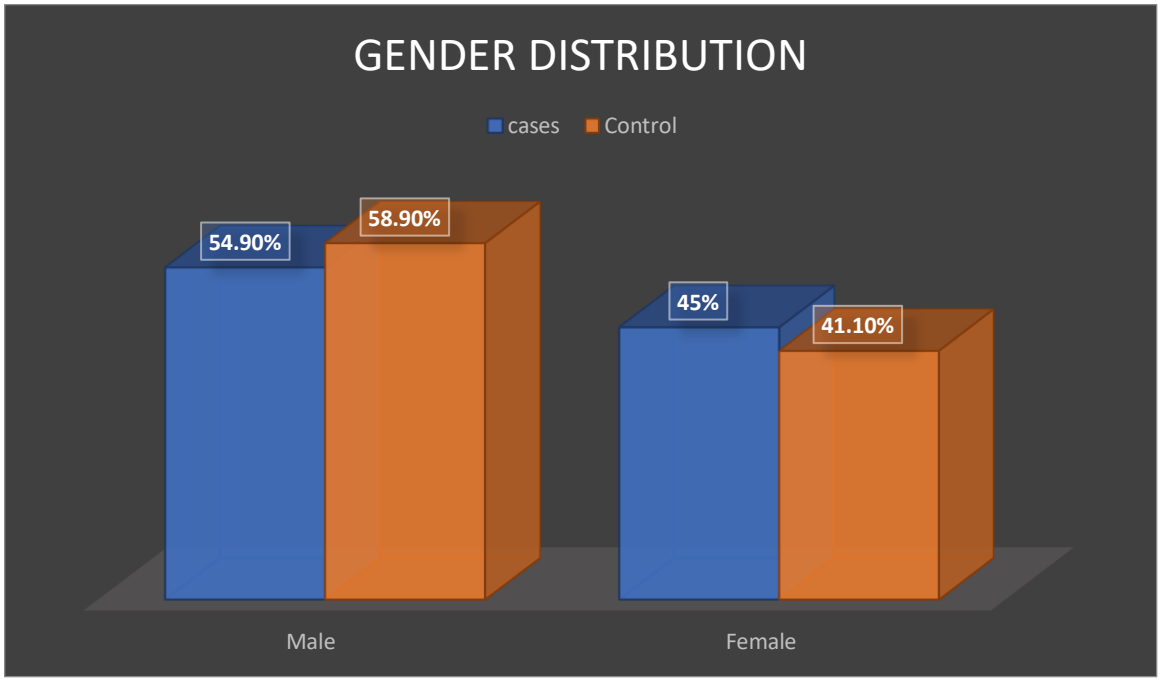

Figure 11: Gender wise distribution of study participants

Table 3: Distribution of study participants according to Past history

Sl No	Past history	Cases n(%)	Control n(%)
1	Present	19(37.3%)	15(26.8%)
2	Absent	32(62.7%)	41(73.2%)
3	**Total**	**51(%)**	**56(100.0%)**

The table presents the distribution of past medical history among study participants. In the case group, 37.3% (n=19) had a notable history, with the majority having conditions such as seizure disorder, TB meningitis, cerebral palsy, and febrile convulsions. In contrast, 62.7% (n=32) had no significant past medical history.

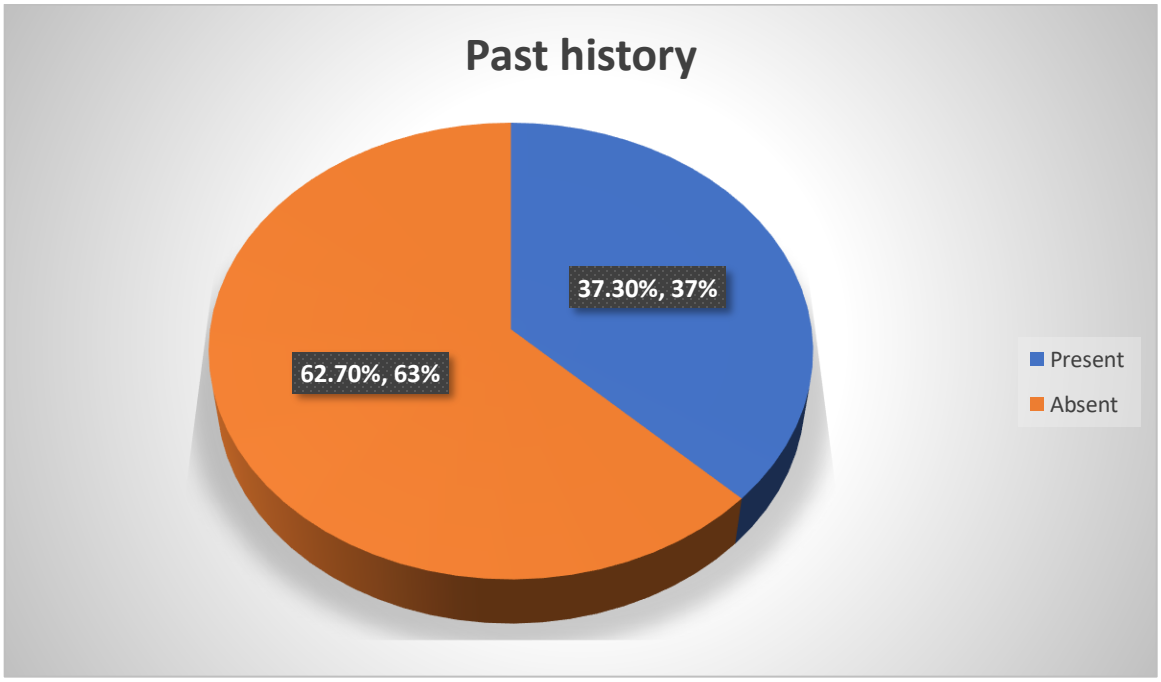

Figure 12: Distribution of study participants according to Past history

Table 4: Distribution of Total leucocyte count among study participants

Sl No	Total Leucocyte count	Cases Mean (SD)	Control Mean (SD)
1	Mean	19487.94	16698.96
2	Standard Deviation	11396.750	18930.845

3	Range	57560	139030
4	Minimum	3890	970
5	Maximum	61450	140000

The table presents the mean and standard deviation of the total leukocyte count in the case and control groups. The mean leukocyte count was 19,487.94 (SD: 11,396.750) in the case group and 16,698.96 (SD: 18,930.845) in the control group. These findings are illustrated in the line diagram.

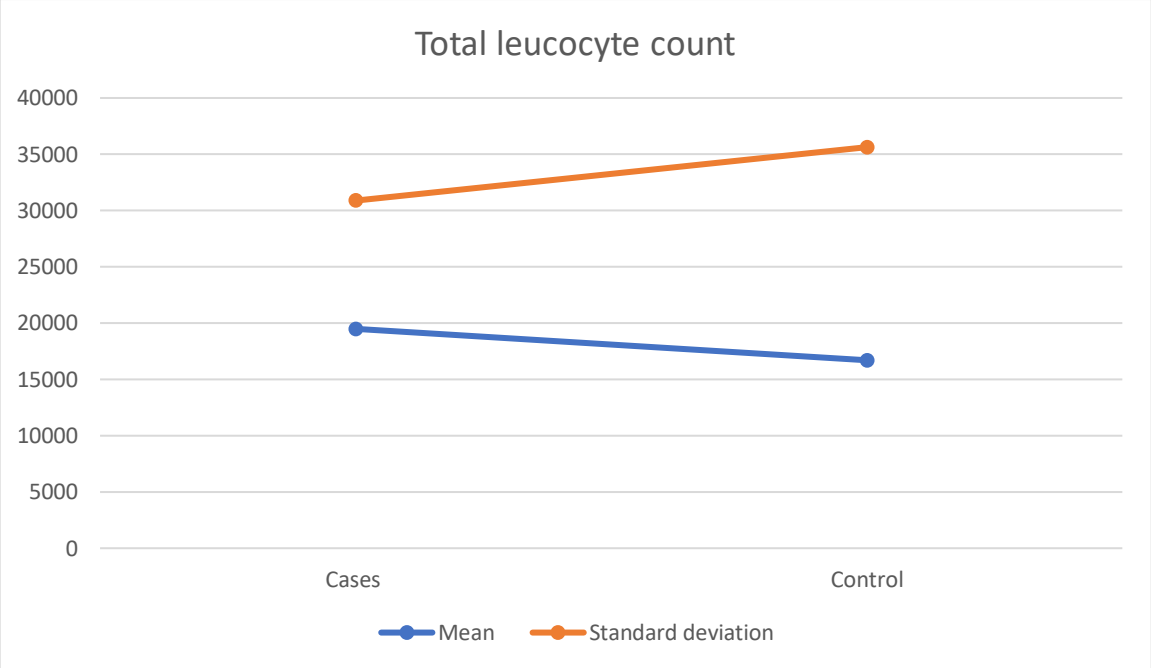

Figure 13 : Distribution of mean and Standard deviation of Total leucocyte count among study participants

Table 5 : Distribution of mean and standard deviation of Platelet count among study participants in both the groups

Sl No	Platelet count	Cases Mean (SD)	Control Mean (SD)
1	Mean	194861.37	209671.79
2	Standard Deviation	137662.431	146883.998
3	Range	765000	576700
4	Minimum	10000	10000
5	Maximum	775000	586700

The table presents the mean and standard deviation of platelet count among study participants. In the case group, the mean platelet count was 194,861.37 (SD: 137,662.4), while in the control group, it was 209,671.79 (SD: 146,883.9).

Table 6 : Distribution of Lactate level among study participants in both case and control group

Sl No	Lactate level on admission	Cases Mean (SD)	Control Mean (SD)
1	Mean	7.06	1.71
2	Standard Deviation	2.130	0.929
3	Range	7	4

4	Minimum	5	0
5	Maximum	12	4

The table presents the lactate levels among study participants. Participants with lactate levels >4 mmol/L were classified as cases, while those with <4 mmol/L were considered controls. The mean lactate level was 7.06 in the case group and 1.71 in the control group. These findings are illustrated in a line diagram.

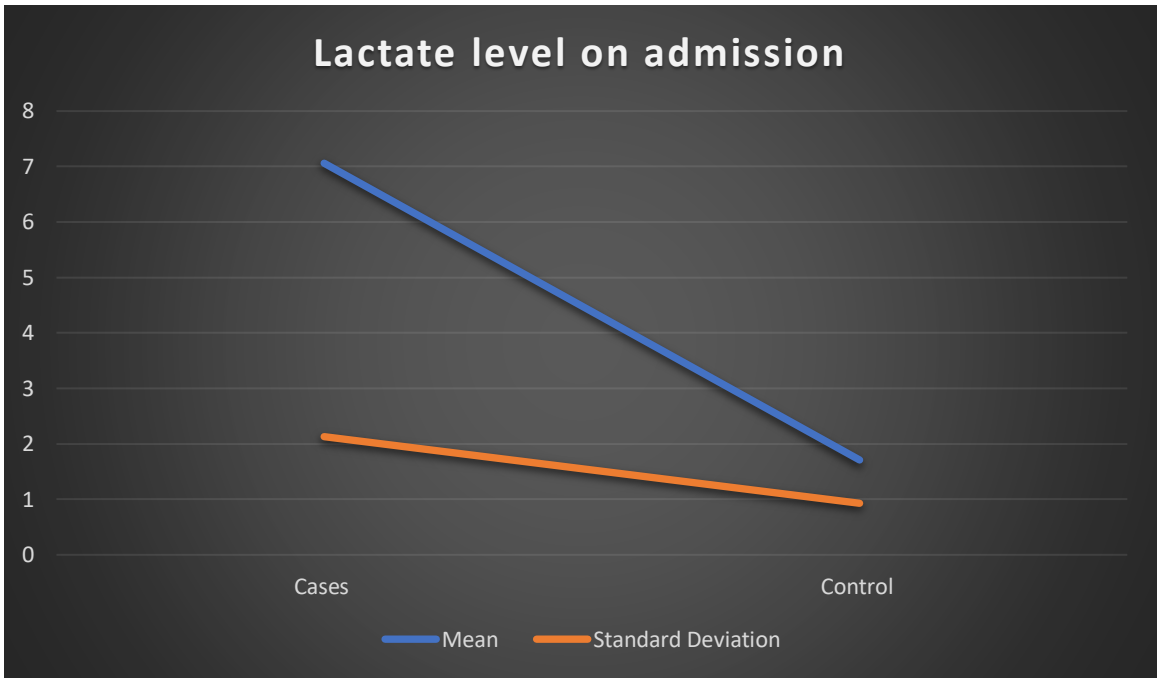

Figure 14: Distribution of Lactate level among study participants in both case & control group

Table 7: Distribution of Lactate level among study participants in both case and control group

Sl No	Lactate level	Cases Mean (SD)	Control Mean (SD)
1	Lactate level at 6^{th} hours	3.96(2.99)	1.64(1.15)
2	Lactate level at 12^{th} hours	3.63(3.14)	1.71(1.85)
3	Lactate level at 15^{th} hours	2.63(2.154)	1.54(1.67)
4	Lactate level at 24 hours	2.61(2.93)	1.74(2.33)

The table presents the mean and standard deviation of lactate levels at the 12th, 15th, and 24th hours among cases and controls. These findings are visually represented in a bar diagram.

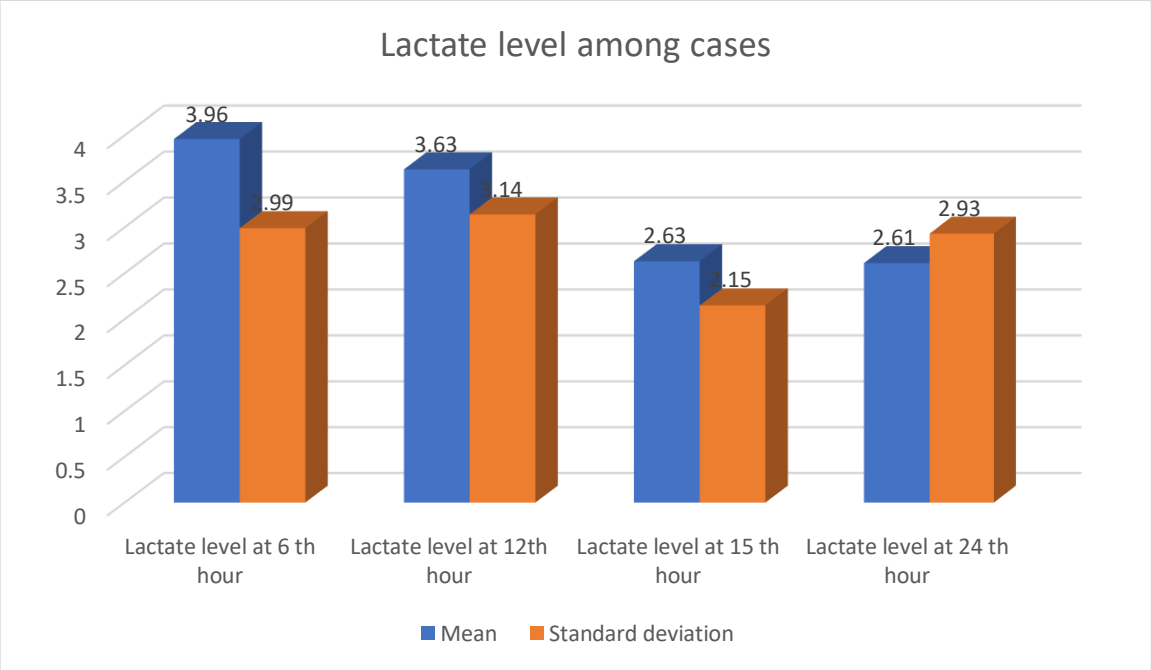

Figure 15: Distribution of Lactate level among study participants in both case and control group

Table 8 : Distribution of Lactate clearance value among study participants in both case and control group

Sl No	Lactate clearance	Cases n(%)	Control n(%)	P value
1	<10%	4(7.8%)	14(25%)	<0.005
2	>10%	47 (92.1%)	42(75%)	
3	Total	51 (%)	56(100%)	

χ^2=5.5,df-1

The table presents the lactate clearance among study participants in the case and control groups. In the case group, 92.1% (n=47) had lactate clearance >10%, while 7.8% (n=4) had lactate clearance

<10%. In comparison, the control group showed 75% (n=42) with lactate clearance >10% and 25% (n=14) with lactate clearance <10% and this found to be statistically significant .These findings are illustrated in a bar diagram.

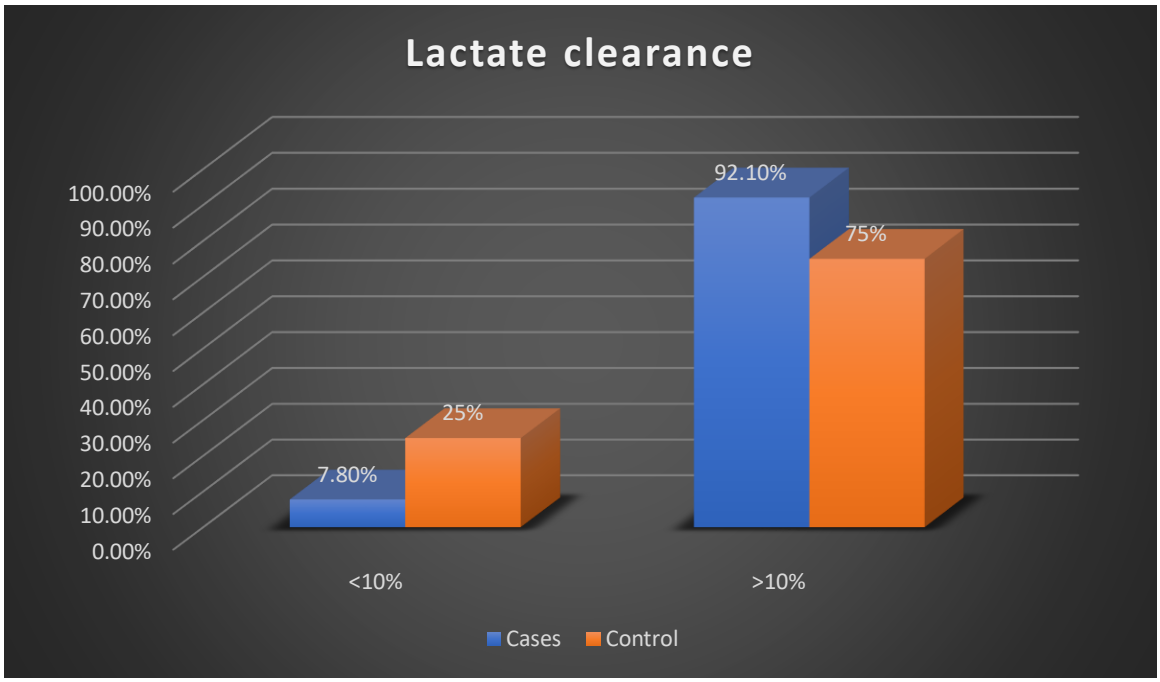

Figure 16: Distribution of Lactate clearance value among study participants in both case and control group

Table 9: Distribution of CRP value among study participants in both case and control group

Sl No	CRP value	Cases n(%)	Control n(%)	P value
1	Normal	6(11.7%)	33 (58.9%)	**<0.0001**
2	Mild elevation	21 (41.1%)	15 (26.7%)	
3	Moderate Elevation	24 (47.05%)	8 (14.2%)	
4	Total	51(100%)	56(100%)	

χ^2=27.58,df-1

The table presents the CRP levels among study participants. In the case group, 47.5% (n=24) had elevated CRP levels, whereas in the control group, only 14.2% (n=8) had moderate CRP levels. These findings are illustrated in a bar diagram and this value found to be significant.

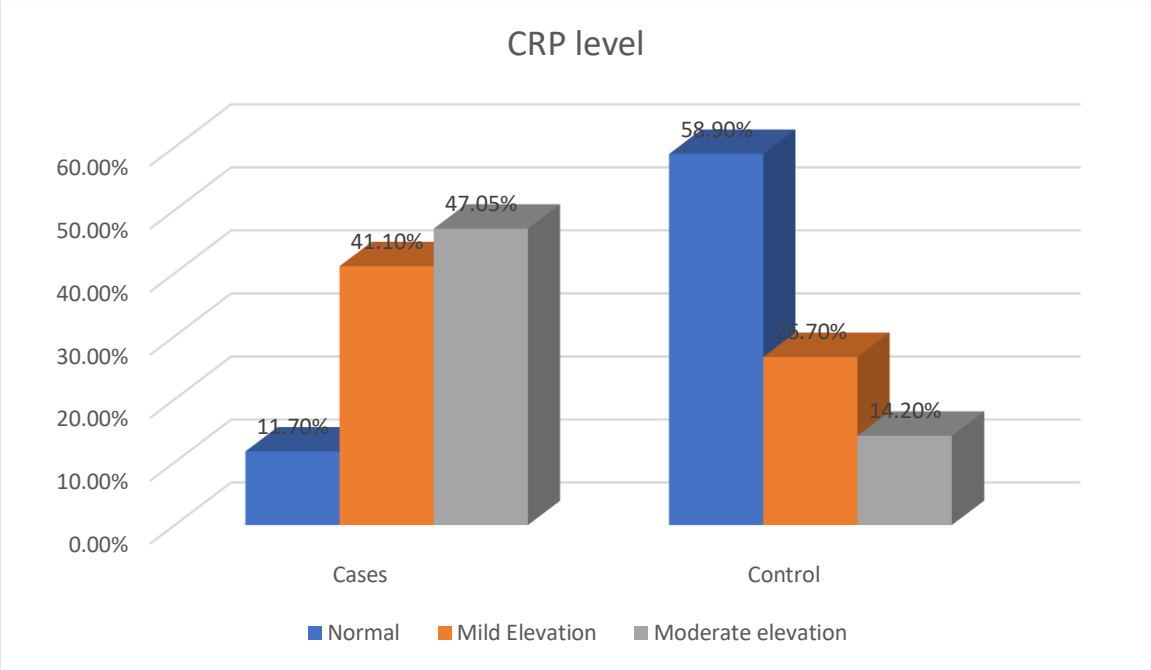

Figure 17 : Distribution of CRP value among study participants in both case and control group

Table 10: Distribution of Outcome variables among study participants in both case and control group

Sl No	Outcome	Cases n(%)	Control n(%)	P value
1	Discharged	39(76.4%)	53(94.6%)	**<0.003**

2	Death	12(23.5%)	3(5.4%)	
3	Total	51(100%)	56(100%)	

χ^2=7.22,df-1

The table presents the distribution of outcome variables among cases and controls. The majority of participants were discharged in both groups, with 76.4% (n=39) in the case group and 94.6% (n=53) in the control group. However, the mortality rate was higher among cases at 23.5% (n=12) compared to 5.4% (n=3) in the control group and this found to be significant

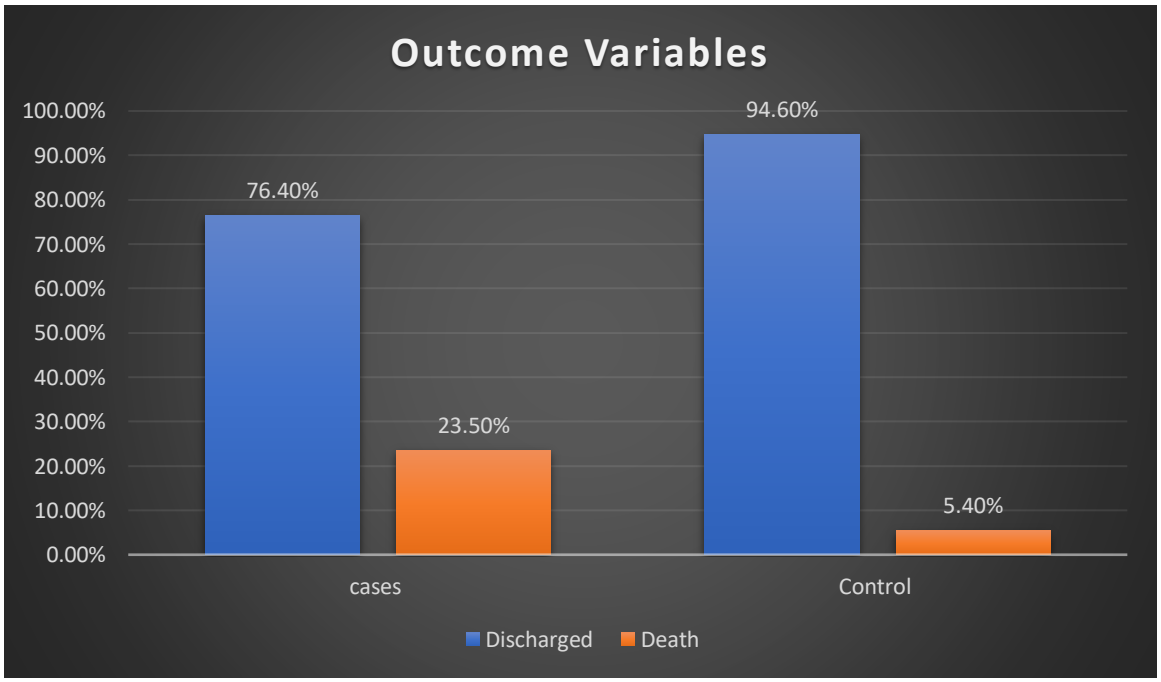

Figure 18: Distribution of Outcome variables among study participants in both case and control group

Table 11: Association between lactate level on admission and Outcome variables

Sl no	Lactate on admission	Outcome		P value
		Discharged	Death	
1	>4	39(42.3%)	12(80%)	**0.007**
2	<4	53(57.6%)	3(20%)	
3	Total	92(100%)	15(100%)	

χ^2=7.31,df-1

The table presents the association between lactate levels and outcome variables among study participants. Chi-square test analysis revealed a significant association between lactate levels and outcomes.

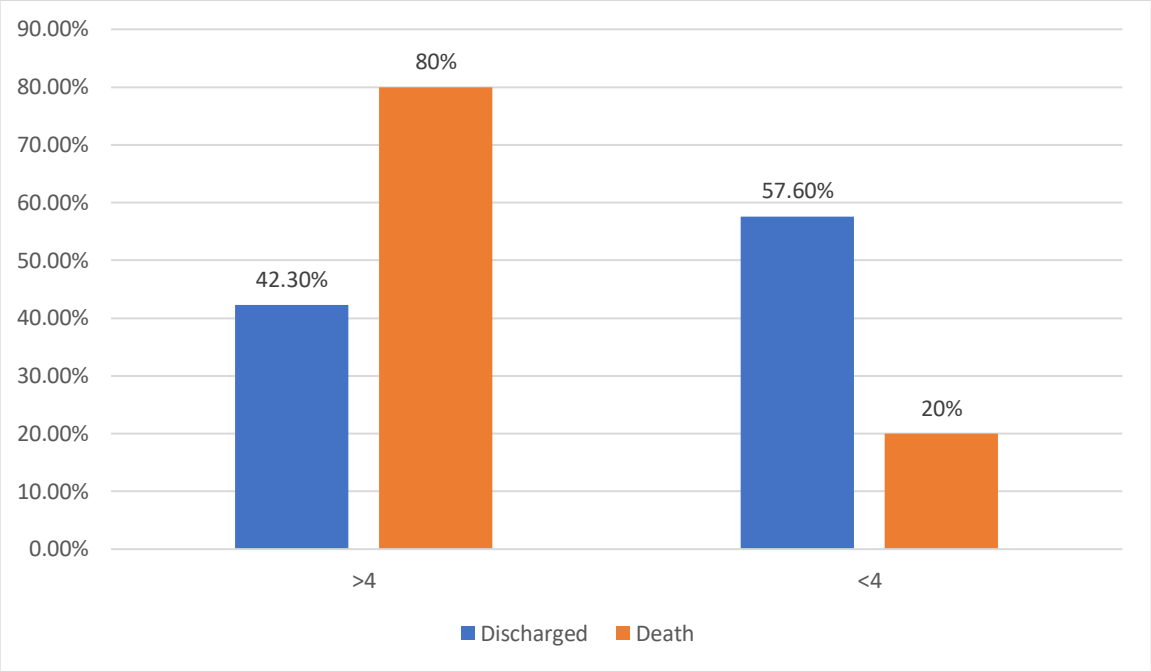

Figure 19: Association between lactate level on admission and Outcome variables

Table 12: Association between lactate clearance on admission and Mean Lactate value

	Lactate clearance		P value
	<10%	>10%	
Lactate level at admission	8.17(2.7)	7.0(2.5)	0.375
Lactate level at 6th hour	4.9(3.17)	3.8(2.9)	0.64
Lactate level at 12th hour	9.2(3.0)	3.0(2.7)	**0.0005**
Lactate level at 15th hour	4.1(5.0)	2.2(1.9)	0.104
Lactate level at 24th hour	9.5(3.4)	2.3(2.4)	**0.001**

The table presents the association between the mean and standard deviation of lactate levels and lactate clearance among cases. The lactate levels at admission, 6th hour, and 15th hour showed no

significant association, whereas the lactate levels at the 12th and 24th hours were significantly associated.

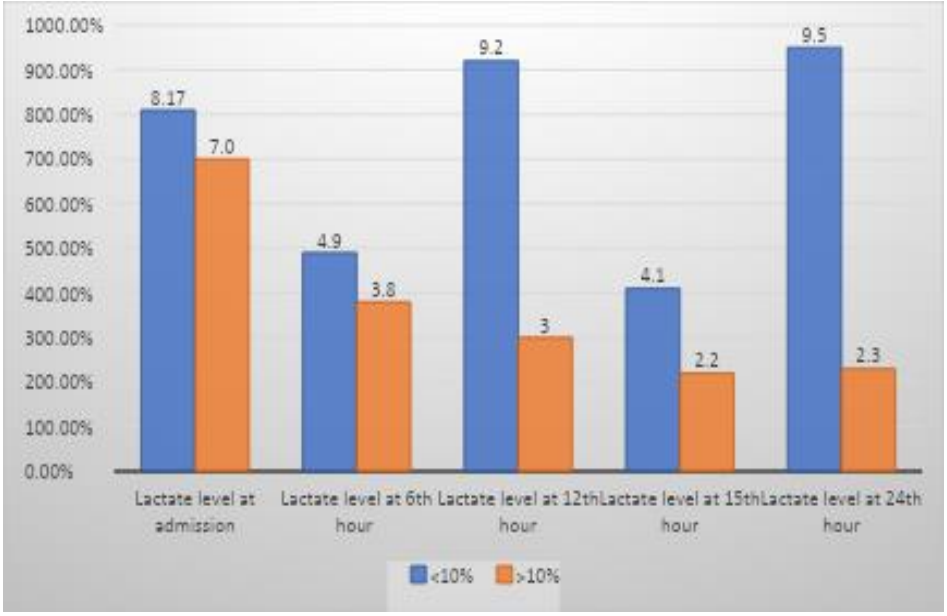

Figure 20: Association between lactate clearance on admission and Mean Lactate value

Table 13: Association between lactate clearance on admission and Outcome variables among both the groups

Sl no	Lactate clearance	Outcome		P value
		Discharged	Death	0.353
1	<10%	15(16.3%)	3(20%)	
2	>10%	77(83.6%)	12(80%)	
3	Total	92(100%	15(100%)	

χ^2=0.4819,df-2

This is the table showing the association between Lactate clearance and Outcome variables among study participants and found that there is no statistical significance between these two on applying Chi-square test

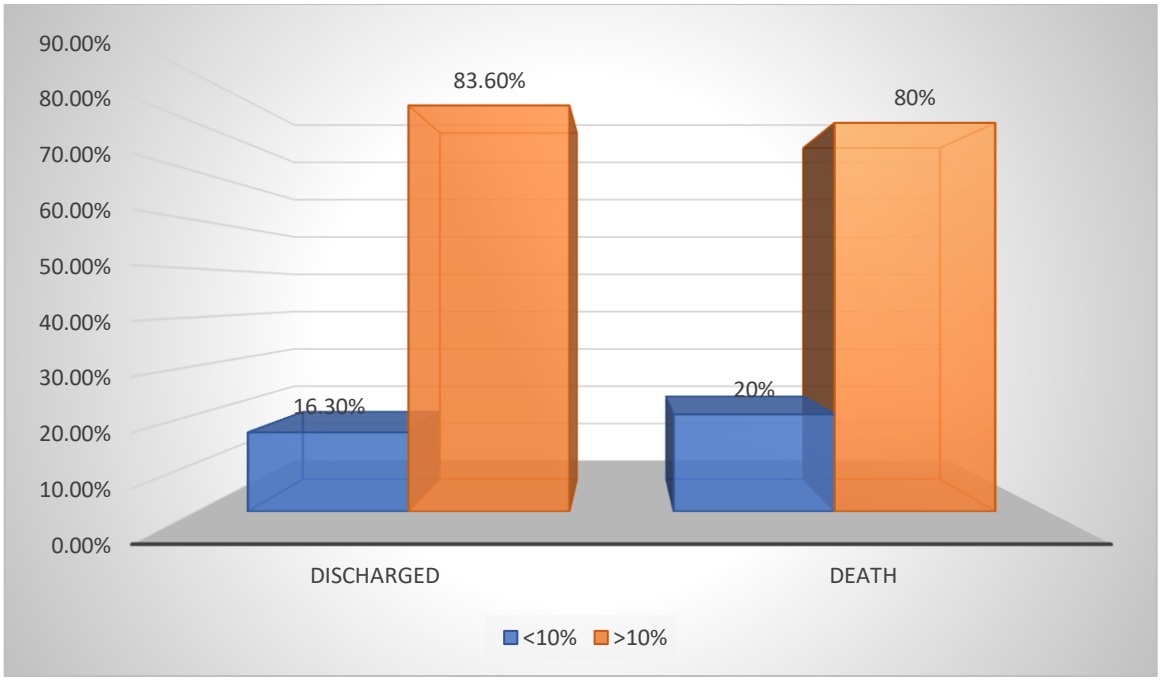

Figure 21: **Association between lactate clearance on admission and Outcome variables among both the groups**

Table14: Association between Lactate clearance and outcome variables among cases

Sl no	Lactate clearance	Outcome		P value
		Discharged	Death	0.194
1	<10%	2(5.14%)	2(16.71%)	
2	>10%	37(94.9%)	10(83.3%)	
3	Total	39(100%)	12(100%)	

χ^2=1.6,df-1

This is the table showing the association between Outcome variables and lactate clearance among cases groups and found that there is no statistically significant between these two and p value found to be >0.005

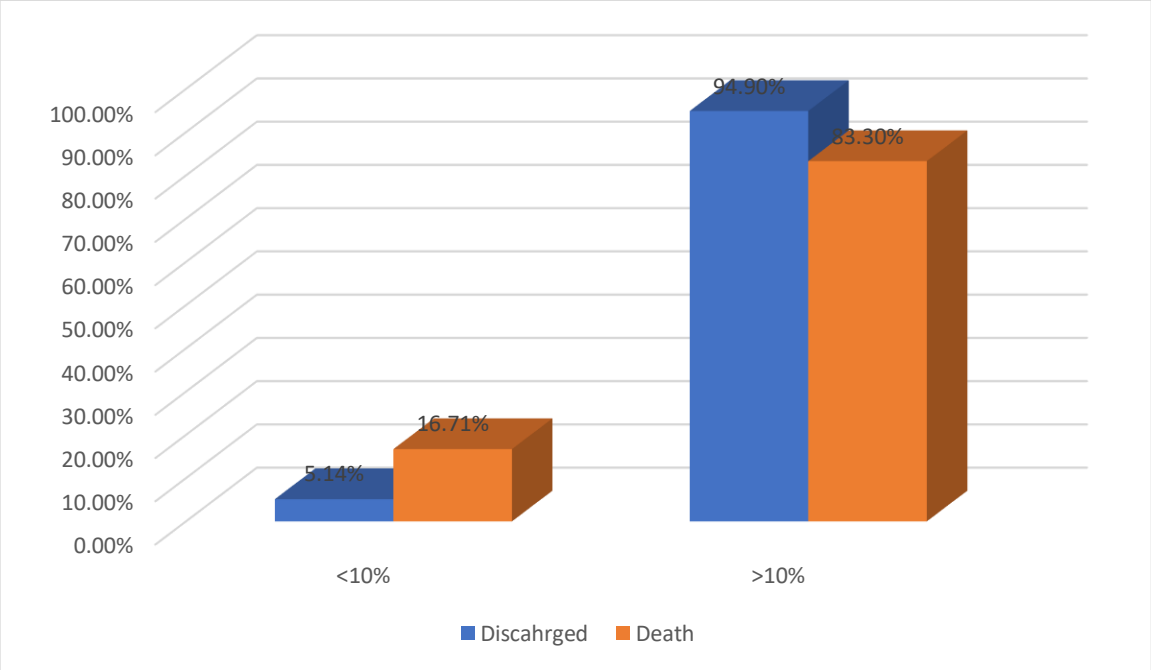

Figure 22: Association between Lactate clearance and outcome variables among cases

Table 15: Distribution of study participants according to Clinical symptoms

Sl No	History	Cases n(%)	Control n(%)
1	ACUTE GE	5(9.8%)	4(7.1%)
2	CONVULSIONS	11(21.6%)	17 (30.3%)
3	OTHERS	3(5.9%)	**3** (5.3%)
4	PNEUMONIA	16(31.4%)	**19** (33.9%)
5	SHOCK	15(29.4%)	**12** (21.4%)
6	UTI/jaundice/epistaxis	1(2.0%)	**2** (3.5%)
7	Total	51(100.0%)	**56(100%)**

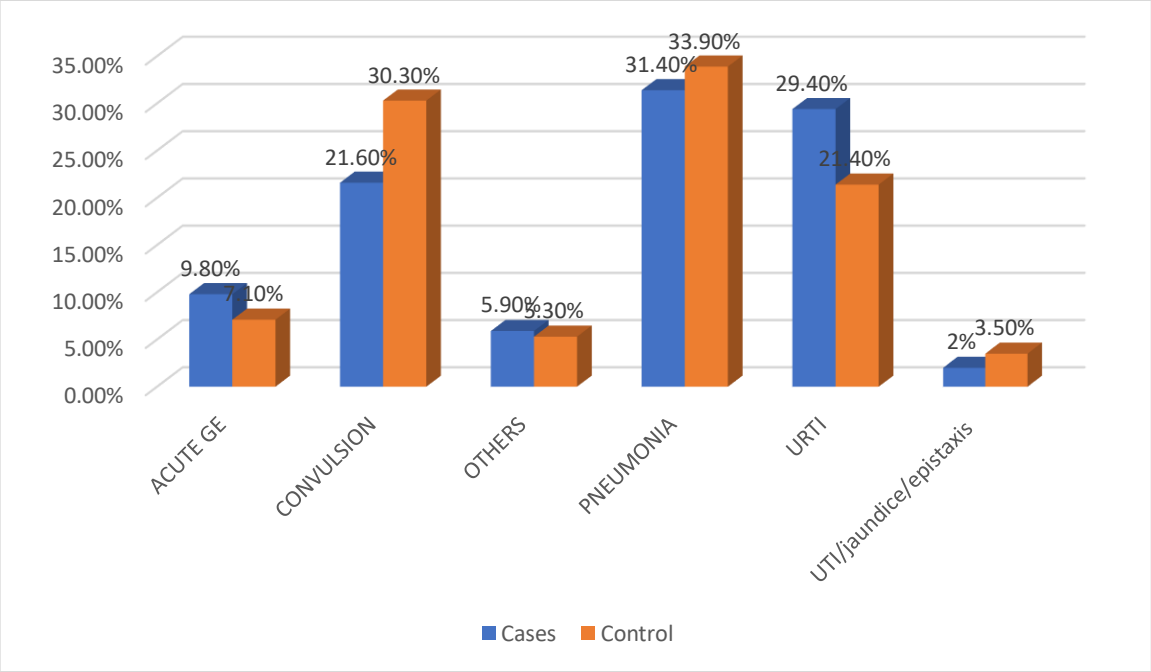

Figure 23: Distribution of study participants according to Clinical symptoms

Table 16: Association between Clinical symptoms and outcome variables among cases

Clinical symptoms	Outcome		P value
	Death	Discharge	
Acute GE	0	5(12.8%)	**0.031**
Seizure disorder	4(33.3%)	7(17.9%)	
Pneumonia	4(30%)	10(25.6%)	
Shock	2(20%)	15(38.5%)	
UTI	0	1(2.6%	
Others (RTA/SNAKE	2(16.7%)	1(2.6%)	

EVENOMATION /SCORPIAN EVENOMATION)			
Total	12(100%)	39(100%)	

This table presents the association between Clinical symptoms and outcome variables amongst the cases which found to **be significant.**

Table 17: Association between Clinical symptoms and lactate clearance among cases

Clinical symptoms	Lactate clearance		P value
	<10%	>10%	
Acute GE	0(%)	5 (10.6%)	0.390
Seizure disorder	1 (25%)	10 (21.3%)	
Pneumonia	0 (%)	16 (34%)	

URTI	2 (50%)	13 (27.7%)	
Shock (septic shock /refractory shock)	6(30%)	1 (2.1%)	
Others	1(25%)	2 (4.3%)	
Total	4(100%)	47(100%)	

This table presents the Clinical symptoms and lactate clearance which was found to be **significant.**

DISCUSSION

A study titled "*SERUM LACTATE LEVEL AS A PREDICTOR OF OUTCOME IN PATIENTS WITH SUSPECTED SEPSIS ADMITTED IN PEDIATRIC INTENSIVE CARE UNIT"* was conducted at our PICU, at Shri BM Patil Medical College Hospital and Research Centre, Vijayapura which included suspected sepsis cases that fulfilled the inclusion and exclusion criteria. Minimum of 100 cases were studied, with the objective to determine the blood lactate levels in patients with sepsis admitted to

pediatric intensive care unit and to correlate with their hospital outcomes, to analyse the correlation between initial serum lactate levels and the severity of sepsis (e.g.: sepsis, severe sepsis, septic shock)

The present study showed that out of 107 study participants, 47% were >5 years of age, followed by 39.2% in the 1-5 years age group while 13.7% belonged to < 1 year age. Gender wise 54.9% were males while females were 45%.

In the case group, 37.3% (n=19) had a notable history, with the majority having conditions such as seizure disorder, TB meningitis, cerebral palsy, and febrile convulsions. Study by **Kanna et al** revealed Pneumonia as the leading cause of septic shock illness which occurred in 73.3% of patients.[7] Additionally **Halden et al** found that males represented the major demographic in both septic shock and control groups with 71% and 80% respectively.[28]

Lactate level

Our research examined the predictive role of serum lactate readings in pediatric sepsis patients who needed intensive care at the Pediatric Intensive Care Unit (PICU). Patients in the case group displayed a statistically elevated mean lactate value of 7.06 mmol/L (SD: 2.130) at admission when compared to controls who averaged 1.71 mmol/L (SD: 0.929). The case group patients exhibited decreasing trend of lactate levels based on multiple measurements at 6-hour intervals starting from 3.96 mmol/L (SD: 2.99) up to 3.63 mmol/L (SD: 3.14) at 12 hours then 2.63 mmol/L (SD: 2.13) at 15 hours before reaching 2.61 mmol/L (SD: 2.93) at 24 hours.

Studies backed by literature support the use of both first-time and ongoing lactate tests as diagnostic markers in pediatric sepsis cases. A similar study conducted by **Jat et al.** demonstrated that survivors of sepsis displayed lower blood lactate values during admission as well as at 12 and 24 hours apart measurements. The mortality risk increased significantly with each rise in blood lactate levels

exceeding 5 mmol/L at specific time points starting from PICU admission up to 3 hours followed by 12 hours and then another rise at 24 hours.[7]

Another similar study by Patriawati et al conducted to determine blood lactate levels in predicting sepsis mortality. The research determined blood lactate levels exceeding 4 mmol/L during admission and 24 hours as strong predictors of patient mortality because they resulted in relative risk calculations of 2.9 and 4.92 respectively. The risk of mortality increased substantially when patients experienced less than 10% lactate clearance up to 24 hours. [29]The baseline lactate measurement above 36 mg/dL (4 mmol/L) in children with suspected sepsis linked strongly to 30-day mortality according to **Scott et al**. The measurements of lactate concentrations higher than 36 mg/dL showed a 20% sensitivity rate in predicting mortality within 30 days with a specificity level of 92.3%. [28]

The mean initial lactate levels recorded in our cases exceeded those previously established thresholds bringing attention to the severe sepsis state in our sample group. Lactate levels demonstrated a decreasing trend during the observation period probably because of successful therapeutic methods and enhanced tissue blood flow. Studies show that patients with both constant elevations of lactate levels or poor lactate clearance function experience mortality risks which demonstrates why repeated lactate monitoring carries significant importance.[50]

A lactate clearance of more than 10% occurred in 92.1% (n=47) of case group patients but only 7.8% (n=4) demonstrated clearance below 10%. The control group presented lactate clearance satisfactory to 75% (n=42) versus 25% (n=14) who experienced inadequate clearance that reached statistical significance. The study showed that lactate clearance introduces valuable prognostic information when it's about managing pediatric septic patients.

Our research findings confirm existing studies, about how lactate clearance predicts death rates in young children experiencing septic shock. A Similar study by **Munde et al**. established that patients who did not achieve more than 30% lactate clearance at 6 hours ended up with 90% mortality yet those with better clearance rates died at 8.5%. A clearance threshold less than 30% had an accuracy of 75% in mortality prediction along with 97% in test specificity. [51]

Lactate clearance demonstrated a statistically significant difference between survivors compared to no survivors at 17.9% ± 39.9% vs. -23.2% ± 62.7% ($P < 0.0001$) according to **Choudry et al**. The evaluation of a 24-hour lactate clearance below ten percent achieved a sensitivity rating of 78.7% and specificity of 72.2% and demonstrated a 83.1% as predictive value for death. A lack of sufficient lactate clearance to 10% or better increased mortality possibilities (confirmation ratio +2.83 with 95% confidence interval 1.82 to 4.41). [52]

The mortality rate rose to 90% among patients who failed to reach a 30% lactate clearance within 6 hours but survivors achieved 8.5% survival according to **Munde et al.** The predictive threshold achieved 75% sensitivity and 97% specificity for mortality prediction. [51]

Among study participants 76.4% (n=39) in the case group received hospital discharge while 94.6% (n=53) in the control group received discharge. The case group experienced statistically higher mortality compared to the control group with sample sizes of 12 patients representing 23.5% mortality rate versus 3 patients and 5.4% mortality rate.

A large systematic review with meta-analysis included data from 94 studies involving 7,561 patients which showed the expected death rates to be 19.3% for developed countries and 31.7% for developing countries. Higher sepsis severity and younger patient age groups further increased the risk of mortality, according to this research. [53]

Another similar study conducted in southern Ethiopia for neonatal sepsis reported that 25.1% of patients with severe sepsis passed away. The study also stated that poor feeding, respiratory distress, gestational age under 37 weeks and convulsions further increased the mortality risk rate[54.]

Our research evaluated the paediatric sepsis patients through their lactate clearance assessment into two groups, defined by their clearance percentage levels whether lower or higher than 10%. Lactate levels proved different between groups starting from the 12th hour after admission with p-values reaching 0.0005 and 0.001 at the 12th and 24 hours intervals respectively.

Studies have proven that the predictive quality of lactate clearance defines paediatrics patient outcomes in sepsis cases. Research conducted by **Scott et al.** revealed that children with suspected sepsis who had lactate values above 36 mg/dL (approximately 4 mmol/L) showed a strong connection to mortality rates within a month. Detection of elevated lactate levels above 36 mg/dL provided clinicians with a 20% risk assessment of short-term mortality outcomes with a 92.3% degree of accuracy for patient survival. Measuring lactate at different points presents an essential technique for evaluating patient outcome [28.]

Results from **Jat et al.'s** research indicated that no survivors demonstrated sustained increased blood lactate levels during admission periods and at 12 and 24 hours intervals. The odds ratios for mortality increased consistently with rising lactate values more than 5 mmol/L during the first 3 hours and subsequently at 12 hours and 24 hours of PICU admission. The necessity of performing multiple lactate tests emerges as a critical factor for forecasting pediatric sepsis treatment results [7.]

Our research showed that among subjects with slow lactate clearance under 10% only 16.3% (n=15) were discharged from hospital but 20% (n=3) perished from the condition. Among patients who

cleared lactate at a rate above 10%, 83.6% (n=77) survived and were discharged from hospital while mortality occurred in 80% (n=12) of the cases. Lactate clearance rates failed to establish a statistical link to patient outcomes in our study sample based on the obtained p-value of 0.353.

These study results oppose previous research that highlights the predictive power of lactate clearance rates in young hospitalized patients under critical care. **Munde et al.** conducted research showing that pediatrics patients whose 6-hour lactate clearance reached under 30% experienced a 90% mortality rate while those achieving above 30% achieved only an 8.5% mortality rate. A survival prediction analysis showed that this threshold identified deceased patients with 75% accuracy as well as 97% accuracy for patients who lived. This pattern demonstrates early lactate clearance's essential part in patient prediction rates. [51]

A study led by **Sandal and colleagues** looked into a group of 172 patients. Sadly, 44 of them passed away. The survivors had lower median lactate levels upon admission, coming in at 4.4 (with a range of 3.1) compared to 5.75 (with a range of 7.7) for those who didn't survive ($p = 0.002$). Those who died (11.7%) had markedly lower lactate clearance at the 6-hour mark than those who made it through, with rates dropping from 36.7 ($p = 0.001$). When lactate clearance was less than 20.7% at 6 hours, it indicated a higher risk of mortality. Both the lactate levels and lactate clearance values were considerably associated with death ($p < 0.05$). There was also a bit of a positive link between the PRISM-IV percentage and the outcomes.[55]

Strengths of the study

1. A prospective research design monitored lactate levels and patient results as they occurred in real time.

2. Laboratory technicians performed objective serum lactate assessments as a part of measuring results that linked patient's progress to diagnostic markers.

3. The study underlines serum lactate clearance as a vital tool for detecting sepsis outcomes that provides early risk assessment abilities for patients.

4. The study demonstrates how lactate level surveillance serves as a valuable prognostic measure for pediatric sepsis treatment thus enabling physicians to make proper early interventions.

5. The researched number of 107 cases met the requirement for acceptable statistical analysis.

6. Early intervention and treatment modifications became possible because the study proved that high-risk patients could be properly distinguished by monitoring their lactate clearance levels between 12 to 24 hours.

7. Quantitative data measurement of lactate clearance along with its outcome associations proved effective in minimizing human subjectivity.

8. The study used precise defined criteria while selecting participants which guaranteed similar research conditions across subjects while eliminating potential bias from selection regulations.

9. Time-specific lactate measurement required nurses to check blood lactate levels at 12-hour and 24-hour intervals for better sepsis biomarker assessment amongst critically ill children.

10. The research results enable PICU practitioners to use lactate clearance as an indicator which aids in sepsis monitoring throughout PICU management.

11. Mortality and Survival Outcome Data contained unambiguous information about discharge rates and mortality to help practicing clinicians in sepsis management.

12. The research outcomes matched global studies demonstrating better results from increased lactate clearance which strengthens the validity of the investigative findings.

13. The study found that lactate measurements at both 12 and 24 hours differed significantly from baseline values according to statistical analysis done at a p-value less than 0.001 which confirms the need for prompt monitoring for sepsis assessment.

14. Statistical data shows strong importance of lactate clearance for early risk assessment through p=0.0005 and p=0.001 p-values observed during the 12th and 24th hours examination period.

15. The concept of utilizing lactate clearance to identify disease prognosis works across different age groups although the current study involved only pediatric patients.

16. They could identify patients at risk of treatment failure who had lactate clearance levels below 10%, which allowed healthcare providers to tweak their treatment strategies for delicate patients.

Limitations of the study

1. The study was limited to Shri BM Patil Medical College Hospital and Research Centre, which makes it harder to apply the findings broadly across various medical facilities.

2. Results would've been more strong if we could include more than 100 cases, which would have strengthened the statistical power.

3. The analysis only tracked results until patients were either discharged or passed away, missing the chance to check on their health after that period.

4. The study didn't look at other factors that might impact lactate clearance, like comorbidities, history of previous treatments or nutrition status.

5. While the research focused on discharge status and mortality, it didn't consider other treatment variables like how long patients stayed in the hospital or any organ damage.

Recommendations

1. Future studies should aim for larger-scale research across multiple sites to explore the connection between sepsis outcomes and lactate clearance in children.

2. Researchers ought to extend monitoring protocols to see how lactate clearance affects patient's health after they leave the hospital and their overall quality of life.

3. It's important for future studies to conduct multivariate analysis to really nail down the relationship between lactate clearance and outcomes by accounting for any confounding factors.

4. Labs should set specific times to measure lactate levels, from 6 to 48 hours, to track any changes and evaluate the risk of patient's outcomes.

5. Hospital protocols should incorporate lactate clearance monitoring as an early indicator for managing and intervening in pediatric sepsis cases.

6. When it comes to studying pediatric sepsis, we should also look at multiple secondary indicators like ICU stays, ventilator independence, and organ health to get a complete clinical picture.

Conclusion

In this study, the research team figured out that monitoring the serum lactate clearance in PICU patients can really help in predicting their outcomes while dealing with pediatric sepsis. It turns out that patients who had a lactate clearance above 10% had a much better survival and discharge rate, while those below that threshold faced tougher outcomes, with only 16.3% surviving. We definitely need more extensive multicentre studies with larger groups and longer follow-ups since this current study didn't find a strong statistical link to validate lactate clearance as a reliable predictor for pediatric sepsis treatment.

SUMMARY

The study called "Serum Lactate Level as a Predictor of Outcome in Patients with Suspected Sepsis Admitted in Pediatric Intensive Care Unit" was carried out at Shri BM Patil Medical College Hospital and Research Centre, Vijayapura, involving at least 107 pediatric patients who met the inclusion and exclusion criteria. The main goal was to look at the serum lactate levels and find a correlation to their hospital outcomes and also how lactate clearance correlated with the severity of sepsis (sepsis, severe sepsis, or septic shock).

The findings showed that there were clear differences in lactate clearance levels between the two groups: one maintained clearance levels above 10%, while the other stayed below that mark. When the lactate clearance was above 10%, survival rates were impressive at 83.6%, leading to a higher rate of patient discharge. On the flip side, clearance rates below 10% resulted in a major increase in mortality (20%) and poorer patient outcomes. The lactate clearance rates analysed in this study between 12 and 24 hours showed meaningful statistical differences (p-values 0.0005 and 0.001), but there was not a direct link to patient outcomes (p-value = 0.353).

According to this study, measuring lactate clearance within the first 12-24 hours of PICU admission can act as an early indicator of sepsis outcomes. However, the data didn't provide enough statistical support to solely rely on lactate clearance as a predictor for patient outcomes.

This study took a proactive approach with an objective way of measuring lactate and clearly separated the at-risk patients from those who were not, based on their lactate clearance levels. However, the conclusions drawn were somewhat limited due to a smaller sample size, study being conducted at a single centre, and lacking a deeper multivariate analysis along with a short follow-up period.

Overall, the study emphasized that keeping an eye on serum lactate values, along with monitoring the clearance rates, really helps in assessing risk and guiding treatment decisions for pediatric patients with sepsis. To keep up with the dynamics of the medical field, one needs an extensively

thorough multicentric research to confirm whether lactate clearance assessment can really be an effective predictor for the clinical outcomes in pediatric sepsis.

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RESEARCH INFORMED CONSENT FORM

BLDEA's Shri B.M. PATIL Medical College, Hospital & Research Centre,

Vijayapura, 586103

TITLE OF THE PROJECT: **TO DETERMINE BLOOD LACTATE LEVELS IN PATIENTS WITH SUSPECTED SEPSIS ADMITTED IN PEDIATRIC INTENSIVE CARE UNIT**

GUIDE: DR. S.V. PATIL

MD PAEDIATRICS

PROFESSOR

DEPARTMENT OF PEDIATRICS

PG STUDENT: Dr. SHUBHAM KUMAR

POST GRADUATE IN PEDIATRICS

DEPARTMENT OF PEDIATRICS

PURPOSE OF RESEARCH:

The present study will help in determining the “Role of blood lactate levels in patients with suspected sepsis in PICU’’

PROCEDURE:

I do understand that after having obtained a detailed clinical history, thorough clinical examination and relevant investigations, a Case control study of suspected case of sepsis.

RISK AND DISCOMFORTS:

I understand there is no risk involved and that the child may experience some pain and discomforts during the examination. This is mainly the result of the condition, and the procedures of this study are not expected to overemphasize these feelings, which are in association with the regular course of treatment

BENEFIT:

I do understand that my participation in this study will have no direct benefits to me, other than the potential benefit of the research and education.

CONFIDENTIALITY:

I understand that the medical information produced by this study will become a part of hospital records and will be subjected to confidentiality. Any information about sensitive, personal nature will not be a part of the medical record but will be stored in the investigations research file. If any of the data are used for publication in the medical literature or for teaching purpose, no name will be disclosed, and other identifiers such as photographs will be used only with special written permission taken priorly. I also understand that I may visualize the photograph before granting permission

REQUEST FOR MORE INFORMATION:

I understand that I may ask questions about the study at any time; Dr. SHUBHAM KUMAR at the department of Pediatrics is available to answer my questions or concerns. I understand that I will be informed of any significant new findings discovered during the course of the study, which might influence my continued participation. A copy of this consent form will be given to me to keep for careful reading.

REFUSAL FOR WITHDRAWAL OF PARTICIPATION:

I understand that my participation is voluntary and that I may refuse to participate or may withdraw consent and discontinue participation in the study at any time without prejudice. I also understand that Dr. SHUBHAM KUMAR may terminate my participation in the study after he has explained the reasons for doing so.

INJURY STATEMENT:

I understand that in the unlikely event of injury to my baby, resulting directly from baby's participation in this study; if such injury were reported promptly, the appropriate treatment would be available to the baby. But no further compensation would be provided by the hospital understand that by my agreements to participate in this study and not waiving any of my legal rights.

I have been explained about the purpose of the research, the Procedures required and the possible risks to the best of my ability.

Dr. SHUBHAM KUMAR

[Investigator]

DATE :

PARENTS / GUARDIAN CONSENT STATEMENT:

We confirm that Dr. SHUBHAM KUMAR is doing a study "SERUM LACTATE LEVEL AS A PREDICTOR OF OUTCOME IN PATIENTS WITH SUSPECTED SEPSIS ADMITTED IN PEDIATRIC INTENSIVE CARE UNIT"

A prospective COHORT follow up study. Dr. SHUBHAM KUMAR has explained to us the purpose of research and the study procedure. We are willing to give as much as information required for the study and consent for investigations and the possible discomforts as well as benefits. We have been explained all the above in detail in our own language, and we understand the same. Therefore, we agree to give consent for baby's participating as a subject in this research project .

(Parents / Guardian)

DATE:

[WITNESS TO SIGNATURE

B.L.D.E (DEEMED TO BE UNIVERSITY) ಶ್ರೀ ಬಿ.ಎಂ.ಪಟ್ಟಣಶೆಟ್ಟಿ ಮೆಡಿಕಲ್ ಕಾಲೇಜು, ಆಸ್ಪತ್ರೆ ಮತ್ತು

ಸಂಶೋಧನಾ ಕೇಂದ್ರ , ವಿಜಯಪುರ-586103

ಪರಿಣಾಮ ಬಂಧ/ಸಂಶೋಧನೆಯಲ್ಲಿ ಪಾಲ್ಗೊಳ್ಳಲು ಮಾಹಿತಿ ಪಡೆದ್ ಸ್ಮಮತಿ

ನಾನು, ಕೆಳಗಿನವರು_____ಸ್ಥಿಯಿಟ್ಟವರು, ಮಗ/ಮಗಳು/ಪತಿಯ _____

ವಯಸ್ಸು

_____ವರ್ಷಗಳು, ಸಾಮಾನ್ಯವಾಗಿ ನಿವಾಸಿಸ್ಸುವ ಸ್ಥಳ್ದ ಹೆಸ್ರು_____, ಇಲ್ಲಿ

ಹೇಳಿದ್ದಿನೆ/ಫೀಷಿಸ್ಸತ್ತು ಿನೆ ಡಾಕಟರ್ ಹೆಸ್ರು__ಅವರು ಆಸ್ಪತ್ರೆ ಹೆಸ್ರು_____

ಅವರು ನನಗೆ ಪೂರ್ಣವಾಗಿ ಪರೀಕ್ಷಿಸಿದ್ರು ದಿನಾಂಕದಿಲ್ಲ ____ಸ್ಥಳ್ ಹೆಸ್ರು_____

ಮತ್ತು

ನನಗೆ ನನಿ ಭಾಷೆಯಲ್ಲಿ ವಿವರಸ್ಲಾಗಿದ್ ನಾನು ಒಂದು ರೀಗ (ಸಿಥ ತಿ) ಅನುಭವಿಸ್ಸತಿು ದ್ದ ೀನೆ. ಮಂದುವರದು ಡಾಕಟ ರ್ ನನಗೆ ತಿಳಿಸಿದ್ದದ ರೆ ಅವರು ಒಂದು ಪದ್ದ ತಿ/ಸಂಶೀಧನೆ

ನಡೆಸ್ಸತಿು ದ್ದದ ರೆ

ಶ್ೀಷಿಷಕೆಯುಳ್ಳ____ಡಾಕಟ ರ್_____ಮಾಗಷದ್ಶ್ನದ್ಲ್ಲಿ ನನಿ ಪಾಲ್ಗೊ ಳುಳ ವಿಕೆಯನುು ಕಳಿದ್ದದ ರೆ ಅಧಯ ಯನದ್ಲ್ಲಿ .

ಡಾಕಟ ರ್ ನನಗೆ ಇದ್ನುು ಕೂಡಾ ತಿಳಿಸಿದ್ದದ ರೆ ಈ ಕರ ಮದ್ ನಡೆವಲ್ಲಿ ಪರ ತಿಕೂಲ ಫಲ್ಲತಂರ್ಗಳ್ನುು

ಎದುರಸ್ಬಹುದು. ಮೇಲೆ ಹೇಳಿದ್ ಪರ ಕಟ್ಣೆಗಳ್ಲಿ , ಅಧಿಕಾಂವ್ಯು ಚಿಕ್ಷತಿು ಸ್ಬಹುದ್ದದೂು ಅದ್ನುು

ನಿರೀಕ್ಷಿ ಸ್ಲಾಗುತಿು ಲಿ ಆದ್ದ ರಂದ್ ನನಿ ಸಿಥ ತಿಯ ಹಿರದ್ದಗುವ ಅವಕಾವ್ಯಿದ್ ಮತ್ತು ಅಪರೂಪದ್

ಸಂದ್ಭಷಗಳ್ಲಿ ಅದು ಮರರ್ಕಾರಕವಾಗಿ ಪರರ್ಮಿಸ್ಬಹುದು ಹಂದಿದ್ ರೀಗನಿರ್ಧಷರ ಮತ್ತು

ಯಥಾಕ್ಯು ಚಿಕ್ಷತ್ಯು ಮಾಡಲು ಹಂದಿದೂು. ಮಂದುವರದು ಡಾಕಟ ರ್ ನನಗೆ ತಿಳಿಸಿದ್ದದ ರೆ ನನಿ

ಪಾಲ್ಗೊಳ್ಳುವಿಕೆ ಈ ಅಧ್ಯಯನದ ಫಲ್ಲಿತಂಗಳ ಮೌಲ್ಯಮಾಪನದಲ್ಲಿ

ಸಾಹ್ಯಕವಾಗುತ್ತದೆ ದ ಇತರ

ಸಾಮಾನ್ಯ ಪರಿಕರಗಳ ಚಿಕ್ಷತ್ಸೆಗೆ ಉಪಯುಕ್ತ ಉಲ್ಲೇಖವಾಗಿದೆ, ಮತ್ತು

ನಾನು ಅನುಭವಿಸ್ಸವ ರೀಗದಿಂದ

ವಿಮರ್ಶೆ ಅಥವಾ ಗುರ್ಮಾಗೊಳ್ಳುವ ವಲ್ಲಿ ನನಗೆ ಪರಿಣಿತನಾಗಬಹುದು.

ಡಾಕಟರ್ ನನಗೆ ಇದ್ದುದಾಗಿ ಕೂಡಾ ತಿಳಿಸಿದ್ದಾರೆ ನನಗೆ ಎಂದ್ ನೀಡಿದ್

ಮಾಹಿತಿ, ಮಾಡಿದ್ ಪರಿಶೀಲನೆಗಳು /

ಫೀಟೀಗ್ರರ ಫ್ಲಳು / ವೀಡಿಯೀ ಗ್ರರ ಫ್ಲಳು ನನಗೆ ಮೇಲೆ ತ್ರಗೆದುಕೊಳ್ಳಲಾಗುವ ಅನೆವ

ಶೀರ್ಕರು

ರಹಸ್ಯವಾಗಿ ಇಡುವರು ಮತ್ತು ನಾನು ಅಥವಾ ನನಗೆ ಕಾನೂನು ದೃಷಿಟಯಲ್ಲಿ

ಸಂಬಂಧಿತrannu ಹರತ್ತಪಡಿಸಿ

ಇತರ ವಯಕ್ತಿಯಿಂದ್ ಮೌಲ್ಯಮಾಪನ ಮಾಡಲಾಗುವುದಿಲ್ಲ . ಡಾಕಟರ್ ನನಗೆ

ತಿಳಿಸಿದ್ದಾರೆ ನನಗೆ

ಪಾಲ್ಗೊಳ್ಳುವಿಕೆ ಶುದ್ಧವಾಗಿ ಸ್ವಇಚ್ಛಾಪೂರಿತ, ನನಗೆ ಎಂದ್ ನೀಡಿದ್

ಮಾಹಿತಿಯ ಆರ್ಧರದ್ ಮೇಲೆ, ಚಿಕ್ಷತ್ಸೆ /

ಅಧ್ಯಯನದ್ ಸಂಬಂಧದಲ್ಲಿ ರೀಗನಿರ್ಧಾರ, ಚಿಕ್ಷತ್ಸೆಯ ವಿರ್ಧನ, ಚಿಕ್ಷತ್ಸೆಯ

ಫಲ್ಲಿತಂರ್ ಅಥವ ಆ

ಭವಿರ್ಯ ದ್ ಪರ ವ್ಯತಿ ಗಳು ಬಗೊ ಯಾವುದೇ ಸ್ಪ ರ್ಟ ತ್ರ ಕಳ್ಬಹುದು. ಅದೇ ಸ್ಮಯದ್ಲ್ಲಿ ನನಗೆ ತಿಳಿಸ್ಲಾಗಿದ್ ನಾನು ಯಾವುದೇ ಸ್ಮಯದ್ಲ್ಲಿ ಈ ಅಧಯ ಯನದ್ಲ್ಲಿ ನನಿ ಪಾಲ್ಗೊ ಳುಳ ವಿಕೆಯನು ಿ

ನಿಲ್ಲಿ ಸ್ಬಹುದು ನಾನು ಬಯಸಿದ್ರೆ ಅಥವಾ ಅನೆವ ೀರ್ಕರು ಅಧಯ ಯನದಿಂದ್ ಯಾವುದೇ ಸ್ಮಯದ್ಲ್ಲಿ

ನನಿ ನು ಿ ನಿಲ್ಲಿ ಸ್ಬಹುದು.

ಪರ ಬಂಧ ಅಥವಾ ಸಂಶೀಧನೆಯ ಸ್ವ ಭಾವ, ಮಾಡಿದ್ ರೀಗನಿರ್ಧಷರ ಮತ್ತು ಚಿಕ್ಷತ್ಸ್ರ ಯ ವಿರ್ಧನವನು ಿ

ಅಥಷಮಾಡಿಕೊಂಡು, ನಾನು ಕೆಳ್ಗಿನ ಶ್ರ ೀ / ಶ್ರ ೀಮತಿ_____ನನಿ ಪೂರ್ಷವಾದ್ ಪರ ಜ್ಞೆ ಯ

ಸಿಥ ತಿಯಲ್ಲಿ ಹೇಳಿದ್ ಸಂಶೀಧನೆ / ಪರ ಬಂಧದ್ಲ್ಲಿ ಪಾಲ್ಗೊ ಳ್ಳ ಲು ಒಪುಪ ತ್ಸ್ರ ೀನೆ.

ರೀಗಿಯ ಸಿಹ್ನ ಡಾಕಟ ರನ ಸ್ಹ ಸಾಕ್ಷಿ ಗಳು

ANNEXURE - VII SCHEME

OF CASE TAKING

PROFORMA

Name :

Age :

Sex :

Chief complaints :

Past history: significant / not significant

VITALS:

TEMPERATURE- HR -

RR- BP-

SYSTEMIC EXAMINATION:

CVS:

RS:

P/A:

CNS :

Diagnosis:

INVESTIGATION:

COMPLETE BLOOD COUNT

BLOOD CULTURE

DIAGNOSIS :

BIODATA OF GUIDE

NAME:	Dr. S. V. PATIL
DOB:	12/10/1963
EDUCATION:	M.B.B.S 1987-88 JNMC BELGAVI M.D PEDIATRICS -1992 JNMC BELGAVI
KMC REGISTRATION NUMBER:	27589
WORK EXPERIENCE:	UG TEACHER EXPERIENCE - 27 YEARS PG TEACHERS EXPERIENCE- 19 YEARS
MEMBERSHIP :	LIFETIME MEMBER OF IAP No- L/2017/P-1100
PRESENTLY WORKING AS:	PROFESSOR DEPARTMENT OF PEDIATRICS SHRI B.M. PATIL MEDICAL COLLEGE, HOSPITAL, BLDE (Deemed to be University) VIJAYAPURA – 586 103

INVESTIGATOR BIODATA

NAME:	Dr. SHUBHAM KUMAR
QUALIFICATION:	M.B.B.S
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REGISTRATION NO.	156232
ADDRESS FOR CORRESPONDENCE AND	DEPARTMENT OF PEDIATRICS SHRI B M PATIL MEDICAL HOSPITAL RESEARCH CENTRE VIJAYAPURA - 586103.

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SHRI B. M. PATIL MEDICAL COLLEGE, HOSPITAL & RESEARCH CENTRE, VIJAYAPURA

BLDE (DU)/IEC/ 960/2022-23 10/4/2023

INSTITUTIONAL ETHICAL CLEARANCE CERTIFICATE

The Ethical Committee of this University met on **Saturday, 18th March, 2023 at 11.30 a.m. in the CAL Laboratory, Dept. of Pharmacology,** scrutinizes the Synopsis/ Research Projects of Post Graduate Student / Under Graduate Student /Faculty members of this University /Ph.D. Student College from ethical clearance point of view. After scrutiny, the following original/ corrected and revised version synopsis of the thesis/ research projects has been accorded ethical clearance.

TITLE: "SERUM LACTATE LEVEL AS A PREDICTOR OF OUTCOME IN PATIENTS WITH SUSPECTED SEPSIS ADMITTED IN PEDIATRIC INTENSIVE CARE UNIT".

NAME OF THE STUDENT/PRINCIPAL INVESTIGATOR: DR.SHUBHAM KUMAR

NAME OF THE GUIDE: DR.S.V.PATIL, PROFESSOR, DEPT. OF PEDIATRICS.

Dr. Santoshkumar Jeevangi
Chairperson
IEC, BLDE (DU),
VIJAYAPURA

Chairman,
Institutional Ethical Committee,
BLDE (Deemed to be University)
Vijayapura

Dr Akram A. Naikwadi
Member Secretary
IEC, BLDE (DU),
VIJAYAPURA

MEMBER SECRETARY
Institutional Ethics Committee
BLDE (Deemed to be University)
Vijayapura-586103. Karnataka

Following documents were placed before Ethical Committee for Scrutinization.

- Copy of Synopsis/Research Projects
- Copy of inform consent form
- Any other relevant document

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