MODIFIED QUICK SEQUENTAL ORGAN FAILURE ASSESMENT SCORE WITH SERUM PROCALCITONIN AND NEUTROPHIL AND LYMPHOCYTE RATIO FOR PREDICTING THE OUTCOME FOR ACUTE FEBRILE ENCEPHALOPATHY IN CHILDREN

BY DR ANWITA SINHA

Dissertation submitted to

B.L.D.E (DEEMED TO BE UNIVERSITY)

VIJAYAPURA KARNATAKA

In partial fulfilment of the requirements for the degree of

DOCTOR IN MEDICINE IN PEDIATRICS

UNDER THE GUIDANCE OF

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ABSTRACT

Background: Procalcitonin (PCT) level is one of known biomarker in septic diagnosis, but limited studies report its benefit in predicting the outcomes of children with sepsis. Modified qSOFA and Neutrophil to lymphocyte ratio (NLR) are simple biomarkers of inflammation that can be measured in routine hematological examination which role in predicting organ dysfunction remain unclear.

Objective: To assess the accuracy of modified qSOFA score for predicting outcome in acute febrile encephalopathy.

Methods: A prospective observational study at Shri B M Patil Medical College Hospital and Research Centre in Vijayapura aimed to assess the accuracy of modified qSOFA with serum procalcitonin and neutrophil-lymphocyte ratio in predicting outcomes of acute febrile encephalopathy in children. The study involved 60 patients with suspected central nervous system infection admitted to the PICU over a 1.5-year period. Clinical assessments and blood investigations were conducted at the point of arrival.

Results: Among 60 acute febrile encephalopathy children found that 55%had altered sensorium whereas 45% has intact sensorium. The majority of patients had capillary refill time of less than 3 seconds, with high-risk patients accounting for 35% and low-risk patients making up 65%. High-risk patients comprised 58.3% of the total, while low-risk patients made up 41.7%. 85% of patients were discharged, and 15% died. The progression based on the QSOFA score had a significant association with patients' outcomes, with death being significantly

linked to high risk. Among the 9 patients who died, 88.9% were classified as high-risk. High-risk patients required a PICU stay of more than 3 days, while low-risk patients needed a stay of less than 3 days. Deceased patients had higher modified qSOFA score, and qSOFA score compared to discharged patients. A receiver operating characteristic (ROC) analysis was performed to predict outcomes, with the optimal cut-off value for the qSOFA score and modified qSOFA score being ≥2.50 and ≥4.50, respectively, indicating a higher likelihood of mortality.

Conclusion: Modified qSOFA Score and procalcitonin have shown to have superior performance in predicting the outcome of the patients admitted in pediatric intensive care with acute febrile encephalopathy.

Keywords: qSOFA, NLR, PCT, Acute febrile encephalopathy, Children

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ABBREVIATIONS

AESD - Acute Encephalopathy With Biphasic Seizures And Decreased Diffusion

AFE -Acute Febrile Encephalopathy

ANE -Acute Necrotizing Encephalopathy

CC- critical care

CNS-central nervous system

CRP -C-Reactive Protein

CSF- Cerebro-Spinal Fluid

ED- Emergency Department

EEG -Electroencephalography

ESR -Erythrocyte Sedimentation Rate

FS -Febrile Seizures

HFMD -Hand, Foot, and Mouth Disease

HHV-6- Human Herpes Virus 6

HLA -Human Leukocyte Antigen

HSES -Hemorrhagic Shock Encephalopathy Syndrome)

ICU -Intensive Care Unit

KD -Kawasaki Disease

LqSOFA- Liverpool quick Sequential Organ Failure Assessment

MERS - Mild Encephalopathy with Reversible Splenial lesion

MPR- Mean Platelet Ratio

MPV - Mean Platelet Volume

NLR- Neutrophil to lymphocyte ratio

PLT - Platelet Count

RDW - Red Blood Cell Distribution Width

RR-Resiratory Rate

SBP-Systolic Blood Pressure

SIRS- Systemic Inflammatory Response Syndrome

INTRODUCTION

The word "encephalopathy," which translates to "disease of the brain" in Greek, is vague. It's actually more of a clinical condition with a variety of etiopathologies that affect different organ systems and go much beyond the CNS (Central Nervous System). AFE (Acute Febrile Encephalopathy) is the diagnosis made for a kid who presents with fever, altered sensorium, changed cognition or personality, and/or seizures. AFE is a medical emergency that presents diagnostic and treatment challenges for pediatricians.[1] Encephalitis is the term used to describe a subgroup of these individuals who exhibit signs of either infectious or noninfectious brain parenchymal inflammation. Though there has been significant variance in the incidence of various contributory etiologies worldwide and even within a nation, limited data on children shows CNS infections are the most prevalent cause of AFE in India and other developing countries. In cases with AFE, the general character of the clinical symptoms further complicates the clinical prediction of a potential etiology, which might delay the start of the right medication.

There are various scoring systems that can be utilized, however, identification of life threatening infections in children with fever presenting to the emergency department (ED) has been challenging. Despite the existence of various scoring systems, respiratory rate, blood pressure, and mental status are consistently identified as the most reliable indicators of severe illness in both adults and children. There is a pressing need for pediatric ratings because the qSOFA was developed solely for adult populations. Romaine et al. created

and verified a new, modified qSOFA score (Liverpool quick Sequential Organ Failure Assessment [LqSOFA]), and they evaluated how well it performed in predicting admission to critical care (CC) among febrile children who came to the ED (Emergency Department).

The qSOFA score is a bedside assesment that may identify patients with suspected infections who are at greater risk for a poor outcome outside the intensive Care Unit. qSOFA score includes 1 point for each of 3 criteria: (1) respiratory rate ≥ 22 breaths/min, (2) altered mental status, or (3) systolic blood pressure (SBP) ≤ 100 mm Hg. A qSOFA score ≥ 2 is suggestive of sepsis. qSOFA score has proven to have high sensitivity predicting outcomes for severe illness in adults in the emergency department and intensive care unit in adults. However, unlike adults, paediatric patients often exhibit hypotension later in the course of their illness. Clinical scores such as the qSOFA and SIRS scores have been used in early settings , with worsening scores corresponding to greater in-hospital mortality. For patients requiring ICU-level care, the SOFA score has been shown to be the best predictor of in-hospital mortality. Using the prognostic implications from these clinical scores can help with discussing goals of care as recommended by recent surviving sepsis campaign guidelines. Consequently, including additional parameters is necessary to improve the predictive accuracy of qSOFA in predicting critical care admission in febrile children. No studies have been conducted till date; hence, we

conducted this study to assess the accuracy of modified qSOFA with serum procalcitonin and neutrophil and lymphocyte ratios for predicting outcomes in acute febrile encephalopathy in children.

REVIEW OF LITERATURE

A brain pathobiological disorder known as acute encephalopathy in children and adolescence is characterized by fast progression. Diffuse or extensive noninflammatory cerebral edema is the source of central nervous system dysfunction in acute encephalopathy. Experts from eleven academic societies formed a task group that recently created a unified, consensus-based nomenclature for acute cognitive disturbances. They described acute cognition disturbances as a quickly growing pathophysiological brain process that manifests as delirium, subsyndromal delirium, or coma. According to this statement, subsyndromal delirium is a condition that falls between delirium and normal cognition and in which none of the delirium-specific criteria listed in the "Diagnostic and Statistical Manual of Mental Disorders," fifth edition (DSM-5) are satisfied. The impairment of consciousness in acute encephalopathy lasts for 24 hours with 11 points or less on the Glasgow coma scale, according to the Japanese Society of Child Neurology recommendations for acute encephalopathy in infancy and youth. While certain encephalopathy conditions have a clear etiology, others, such as hepatic or uremic encephalopathy or disorders caused by previous viral infection, are multifactorial. The most common cause of ANE (Acute Necrotizing Encephalopathy) and MERS lesion (Mild Encephalopathy with Reversible Splenial lesion) is the influenza virus. The most frequent cause of acute encephalopathy with biphasic seizures

and decreased diffusion, or AESD, is HHV-6. Mycoplasma and bacteria made up 1% and 2% of the total, respectively. The most prevalent syndromes are AESD, MERS, and ANE. Acute encephalopathy instances linked to viruses such as the cytomegalovirus, rhinovirus, and human metapneumovirus have increased in the past several years.[2]

Acute encephalopathy syndrome was shown to have three different prevalence rates: 29% for AESD, 16% for MERS, and 4% for ANE. In 2% of instances, there were additional symptoms, such as HSES (Hemorrhagic Shock Encephalopathy Syndrome). By 2017, the number of cases each year in Japan had risen to 1115.[2] Even though Japan has an annual incidence rate of around 1000 cases, it is far higher than the number of cases of acute encephalopathy in Western nations. A 2012 questionnaire study done throughout the state of Japan revealed that 56.2% of patients with acute encephalopathy recovered fully, 22.1% had mild to moderate sequelae, 13.5% had severe sequelae, and 5.6% died. In Japan, the overall mortality rate from acute encephalopathy is 6%, with 36% of cases resulting in neurologic sequelae. While AESD-related deaths are rare, neurologic consequences are frequent. Both neurologic sequelae and deaths are prevalent in ANE and HSES.

There is little data to diagnose and treat acute encephalopathy, despite the illness's comparatively high morbidity and death rate. Although there has been discussion about virus outbreaks, regional and racial

inequalities, variances in HLA (Human Leukocyte Antigen), and medical level issues, the actual reason for the high prevalence of acute encephalopathy is still unclear. Additionally, a number of illnesses that can impede the diagnosis and treatment of acute encephalopathy include intracranial infections, autoimmune encephalitis, cerebrovascular diseases, traumatic, metabolic, and toxic disorders, as well as the aftereffects of organ failure. All of these conditions manifest as acute impairments of consciousness. Evaluating and understanding the patterns and variances in clinical practice now is a critical first step in driving future research to provide evidence-based treatment and, in the end, lower the mortality linked to acute encephalopathy. This review aims to provide an updated overview of the literature as well as an opinion on the diagnosis and treatment of juvenile acute encephalopathy.

Clinical presentation :

A characteristic of acute encephalopathy is cerebral oedema without inflammation. This pathologic characteristic raises intracranial pressure, which finally results in reduced cerebral perfusion pressure, herniation syndromes, and/or brainstem dysfunction linked to respiratory and circulatory failure brought on by the central nervous system.[2] Many people often experience seizures, which are frequently feverish and

protracted (febrile status epilepticus). A decline in cognitive functioning, developmental regression ordevelpomental stagnation, a decrease in the child's conscious level, and particular localizing characteristics like seizures, ataxia, tremor, or other focal motor symptoms may occur, depending on the child's age. A fever, headache, nausea, fatigue, and loss of appetite are a few examples of systemic symptoms. Every instance of acute encephalopathy, regardless of the underlying etiology, exhibits at least one symptom, which is a changed mental state. The changed mental state may manifest as something subtle that takes time to manifest, such as apraxia or the inability to draw basic pictures, or it may manifest itself clearly and swiftly, resulting in a coma or death in a matter of minutes.[3]

Metabolic problems and hereditary metabolic problems might present as slowly progressing or static clinical symptoms, which are followed by the onset of an abrupt encephalopathic crisis characterized by lethargy, behavioural abnormalities, or gait impairments brought on by fasting or infections. Systematic inflammatory response syndrome can be present in patients who present with a cytokine storm and includes:

(1) bradycardia or tachycardia;

(2) tachypnoea or the need for mechanical ventilation; and

(3) elevated or depressed leukocytes or 10% immature neutrophils.

Excitotoxicity can induce a mild encephalopathy called acute excitotoxic encephalopathy, which is characterized by a loss of consciousness lasting more than twenty-four hours and generally accompanied by seizures, but without a biphasic clinical course. On the other hand, biphasic seizures are used to diagnose AESD. An extended febrile seizure occurs during an early seizure on day 1, and a complicated cluster of partial seizures occurs during a late seizure on days 4-6.

DIAGNOSIS:

Acute encephalopathy can be clinically indicated by a convulsive state or a coma with clear impairment of awareness; nonetheless, it is very straightforward to diagnose acute encephalopathy in these cases. But there are a number of early warning signs and symptoms, and these symptoms might vary. The International Encephalitis Consortium, which suggests the diagnosis of encephalitis and encephalopathy of probable viral or autoimmune origin, notes that a wide variety of clinical symptoms correspond to abnormalities in brain function. One important requirement is a changed mental state. A fever of at least 38 °C (100.4 °F) within 72 hours of the presentation; generalized or partial seizures not entirely attributable to a prior seizure disorder; a new onset of focal neurological findings; a white blood count of at least 5 mm3 in the CSF (Cerebro-Spinal

Fluid); and an electroencephalogram abnormality consistent with encephalopathy and not due to another factor or condition are additional (minor) criteria to support the diagnosis.

For a child with encephalopathy, a clinical assessment and a treatment plan have to be created together. It is best to have a complete history as soon as possible. To identify systemic signs including rash, lymphadenopathy, and hepatosplenomegaly, as well as to localize brain injury and assess early prognostic indicators, a comprehensive neurologic examination should be carried out.[4] Encephalopathy is often diagnosed by means of clinical procedures that record an altered mental state during a physical examination, such as mental status tests, memory tests, and coordination tests. The findings of clinical tests are often used to identify or suspect encephalopathy. The diagnosis is usually made when the altered mental state is linked to actual another systemic problems, such as renal failure, chronic liver disease, anoxia, or a host of other disorders.[5] Blood levels of glucose, ammonia, lactate, and ketone bodies, together with plasma acid-base status, can all be utilized to determine the subtype linked to hereditary metabolic disorders. Based on certain test results at the beginning and/or during the static periods, the final diagnosis is made.

The serum and CSF concentrations of interleukin and inflammatory tumor necrosis factor are significantly elevated in the cytokine storm subtype. Ferritin, serum aminotransferase, pancreatic amylases, creatine kinase, creatinine, and uric acid nitrogen are significantly elevated in patients with hemophagocytic

syndrome and disseminated intravascular coagulation. The excitotoxic crisis subtype should be diagnosed based on clinical evidence, which includes a biphasic pattern of seizures, varying degrees of altered states of consciousness, and distinctive patterns of cerebral flow images obtained with single-photon emission computed tomography and MRI (Magnetic Resonance Imaging).[6]

A common method for identifying and keeping an eye on kids with acute encephalopathy is EEG (Electroencephalography). The development of technology has made long-term bedside EEG monitoring simpler. The benefit of EEG is that it records brain electrical activity, which allows one to study brain function in real time. Certain children who suffer from acute encephalopathy are generally quite sick and unstable. It is possible to monitor EEG even under these circumstances. Recently, there have been several publications on the use of long-term EEG monitoring in critically ill children with diminished awareness, particularly those suffering from acute encephalopathy. On conventional EEG results in children with acute encephalopathy, several studies have been reported. These findings indicate that EEG abnormalities are more prevalent in children suffering from acute encephalopathy. EEG is therefore thought to be helpful in the diagnosis of acute encephalopathy. These EEG abnormalities include paroxysmal discharges, low voltage, periodic lateralized epileptiform discharges, and widespread, unilateral, or localized slowness.[7]

Nonconvulsive status epilepticus in AESD and FIRES/AERRPS (intermittent, latent seizures) has been shown to be detectable by EEG. The ability to distinguish AESD from persistent febrile seizures may be aided by EEG data. AESD is diagnosed in children who have extended seizures, fever, diminished or absent spindles or fast waves, and persistent or recurrent slowing during sleep. In individuals with encephalopathy, brain imaging and EEG may enhance diagnosis and have prognostic value when paired with the clinical picture. In individuals with encephalopathy, isolated persistent slowing of background activity is the most prevalent EEG result. Numerous diseases, both structural and non-structural, are associated with these patterns.

The diagnosis of encephalitis and its differentiation from other forms of encephalopathy depend heavily on the examination of the CSF. Unless contraindicated, lumbar puncture (LP) should be done as soon as feasible in suspected instances of encephalitis. When determining whether or not an LP is safe to execute, clinical assessment should be employed instead of cranial CT (Computed Tomography). Severe edema may be associated with elevated levels of serum albumin quotient and total protein.[8] Elevated concentrations of cytokines and chemokines in serum and CSF might suggest an excessively combative immune reaction. In certain illnesses, pleocytosis may be detected by CSF investigation, whereas it may not be present in others.[8]

Heterogeneous acute encephalopathy syndrome has been treated since 2000 with imaging technologies including CT, MRI, SPECT, PET, and a range of additional neuroradiological techniques. Since its first

definition in the era of neuroradiographic pictures and clinical data generated from imaging, acute encephalopathy has seen major advancements. In cases of acute encephalopathy, fine cerebral edema pictures were visible. Because CT is accessible in most regional centers and has a short imaging time, it is typically the first test ordered when acute encephalopathy is suspected. The following cranial CT abnormalities are indicative of acute encephalopathy:

(1) low-density zones throughout the brain or possibly the entire cerebral cortex;

(2) no discernible difference between the limbic system medulla and the cerebral cortex;

(3) narrowing of the ventricles and the cerebral subarachnoid space surface;

(4) areas of low density: bilateral thalamus (ANE) and unilateral cerebral hemisphere and

(5) narrowing of the brain's surrounding cisterns: swelling of the brainstem.

Quick Sequential Organ Failure Assessment (qSOFA)

When a febrile child arrives at the emergency department, the qSOFA, which has been developed and validated, can be used to assist identify potentially fatal illnesses. Age-adjusted heart rate, breathing rate, capillary filling time , andconsciousness of the patient, voice, pain, and unresponsive state are all included in the qSOFA. Sepsis-related death was the secondary endpoint, and critical care (CC) admission within 48 hours

after ED presentation was the main outcome. The modified qSOFA outperformed the qSOFA in predicting Critical Care admission, with a net reclassification index of 10.4%.Our study demonstrated improved performance of the modified qSOFA over the qSOFA in identifying febrile children at risk for CC admission and sepsis-related mortality. Further evaluation is required in other settings to ensure the effectiveness of the qSOFA in predicting CC admission in febrile children. Further validation is required in other settings to improve the effectiveness of the qSOFA in identifying febrile children at risk for CC admission and sepsis-related mortality.[9]

A study involving 740 febrile infants aged 29 to 60 days found that measuring procalcitonin levels could improve the identification of low-risk infants who may not need intervention. The study used interrupted time series analyses to evaluate the outcomes of lumbar puncture, antibiotic administration, hospital admission, and emergency department length of stay. The results showed that procalcitonin use increased post-pathway implementation, and the proportion of low-risk infants receiving an LP decreased significantly post-intervention. However, there was no immediate level change post-intervention for LP, antibiotics, admission, or ED LOS. Following the intervention, fewer patients received incomplete laboratory test results and more patients were deemed high risk. No severe bacterial infections were overlooked. The probability of LP was dramatically reduced by a normal procalcitonin level. Procalcitonin testing was quickly accepted by

clinicians, and low-risk newborns used fewer resources. Nevertheless, while more infants had laboratory assessments and were categorized as high-risk post-intervention, there was no change in the population's total usage of resources.[9]

Li et al. determined if the neutrophil-to-lymphocyte ratio (NLR) can predict risk of death in severe HFMD (Hand Foot, and Mouth Disease). A retrospective examination of 664 severe HFMD patients revealed that an NLR cutoff value of 2.01 and 2.50 predicted mortality among all 664 severe HFMD and 137 critical HFMD. The multivariate model identified high fever, EV71 infection, fasting glucose, and NLR (>2.01) as independently associated with death risk. Among 137 critical HFMD patients, EV71 infection, fasting glucose, and NLR (>2.50) were associated with death risk. In conclusion, NLR (>2.01) in severe HFMD and NLR (>2.50) in critical HFMD patients may be associated with increased death risk.[10]

Liu et al. investigated the role of NLR (Neutrophil-to-Lymphocyte Ratio), MPV (Mean Platelet Volume), PLT (Platelet Count) ratio, and RDW (Red Blood Cell Distribution Width) in FS (Febrile Seizures) in Chinese children. The study included 249 children with FS and 249 age-matched controls. Prior to treatment, the inflammatory indicators were computed using total blood cell counts. Logistic regression analysis, t-tests, and chi-square tests were used to assess differences in age, gender, and these markers. The best cut-off values for FS risk were 0.0335 for MPR and 1.13 for NLR. On an additive scale, a noteworthy

synergistic interaction between NLR and MPR was discovered. NLR's ideal cut-off value was 2.549, which had 57.5% specificity and 65.9% sensitivity. On the other hand, there were no statistically significant variations between CFS and SFS in terms of average MPR and RDW values. The relationship between white cell subsets and FS risk is further supported by elevated NLR and MPR. The outcomes demonstrated that NLR is a reliable, albeit somewhat restricted, predictor of the difference between CFS and SFS. Additionally, there's a chance that NLR and MPR will work in concert to affect the likelihood of FS.[11]

Ozdemir et al., evaluated the value of changes in NLR (Neutrophil-Lymphocyte Ratio) and PLR (Platelet-Lymphocyte Ratio) as biomarkers in patients presenting with febrile seizures. A retrospective evaluation of 175 febrile seizure patients and 150 healthy children was conducted. The study discovered a statistically significant difference in the NLR value between the febrile seizure group (3.36±3.28) and the control group (1.82±2.21). Without statistical significance, the PLR value was similarly greater in the group experiencing febrile seizures (135.10±80.44) than in the control group (123.43±67.06). Age did not significantly correlate with either the PLR or NLR values. The kind of febrile seizure was the primary factor impacting the NLR and PLR levels in the febrile seizure group. According to the study's findings, NLR and PLR readings can help distinguish between mild and complicated febrile seizures.[12]

Fuji et al., investigated the use of procalcitonin (PCT) as a biomarker in severe infections. Nine pediatric patients with AESD (Acute Encephalopathy with Biphasic Seizures and Reduced Diffusion) were compared with 10 control patients with febrile seizures. The mean PCT concentrations in the AESD and FS groups were significantly different. The CRP (C-Reactive Protein) levels were also significantly different. The PCT to CRP ratios were 27.5 and 3.2, respectively. The sensitivity and specificity in diagnosing AESD using a PCT/CRP ratio of 1.0 were 79% and 100%, respectively. The results suggest that PCT and the PCT/CRP ratio are useful in auxiliary diagnosis of the second stage of AESD, and in AESD, PCT is likely to increase through a different mechanism.[13]

Lee et al., compared serum procalcitonin levels between febrile children with KD (Kawasaki Disease) and those with viral and bacterial infections. The study involved 49 KD patients, 111 viral infections, and 24 bacterial infections. The mean PCT level in the KD group was significantly lower than in the bacterial infection group and insignificantly different from the viral infection group. The mean ESR (Erythrocyte Sedimentation Rate) and CRP levels in the KD group were significantly higher than in the viral and bacterial infection groups. The proportion of patients with PCT levels >1.0 ng/mL was significantly higher in nonresponders to the initial intravenous immunoglobulin treatment than in responders. The study concluded that PCT levels may help differentiate KD from bacterial infections, and a combination of disease markers,

including ESR, CRP, and PCT, may be useful for differentiating between KD and viral or bacterial infections.[14]

Lee et al., evaluated the effectiveness of the procalcitonin (PCT) test in young febrile infants aged 1 to 3 months. The study involved 336 infants from Samsung Changwon Hospital who visited the emergency department or outpatient department between May 2015 and February 2017. The results showed that 11.3% of the infants had a definitive SBI (Serious Bacterial Infection). The mean PCT and C-reactive protein levels were significantly higher in SBI patients compared to non-SBI patients. PCT had lower sensitivity (43.6%) but higher specificity (92.6%) and accuracy (86.9%) than CRP for identifying SBI. The area under the receiver operating characteristic curves (AUCs) for definitive SBI were PCT 77.0%, CRP 80.8%, WBC 56.8%, ANC 67.8%, and PLT 48.1%. The AUCs for definitive SBI were PCT+CRP 85.4%, PCT+WBC 77.2%, PCT+ANC 81.3%, CRP+WBC 80.1%, and CRP+ANC 81.6%. The study concluded that the PCT test or a combination of PCT and CRP tests, is a more accurate and specific biomarker for detecting and ruled out SBIs.[15]

Vellissaris et al., reviewed the role of procalcitonin as a diagnostic and prognostic marker in adult meningitis patients. A PubMed search was conducted to identify relevant publications and bibliographies. The results suggest that serum procalcitonin has a promising role as a biomarker for meningitis assessment. However, data on cerebrospinal fluid’s procalcitonin's role is limited. The study concludes that S-PCT (Serum

Procalcitonin) is a useful tool for evaluating patients with known or suspected central nervous system infections and distinguishing between bacterial and viral meningitis.[16]

AIMS AND OBJECTIVES

Primary Objective

To assess the accuracy of the modified qSOFA score for predicting outcomes in acute febrile encephalopathy

Secondary Objectives

To compare the modified qSOFA with the qSOFA scoring system.

METHODOLOGY

Source of Data

This is a hospital based, prospective observational study conducted in the PICU, Shri BM Patil Medical College Hospital and Research Centre, Vijayapura. The study included consecutive patients with suspected CNS infections admitted to the PICU, who fulfilled the inclusion criteria. The study was conducted for a period of one and a half years, from June 2022 to January 2024.

Methodology

Patients with suspected CNS infection were admitted to the PICU of Shri BM Patil Medical College Hospital and Research Centre, Vijayapura. A written consent form was obtained from all guardians of patients, fulfilling the inclusion criteria and explaining the objective, procedure, and expected outcome in detail before the start of the study. The patients were included in the study based on the inclusion and exclusion criteria mentioned as follows:

Inclusion Criteria

- All children in the age group of 1 month to 16 years who have a fever with neurological symptoms like altered sensorium, seizures, or excessive irritability.

Exclusion Criteria

- Children with CNS malformations.
- Children with hydrocephalus.
- Children with intellectual disability
- Children with cerebral palsy without fever

Sample Size

With an anticipated proportion of qsofa performance to the modified qSOFA scoring system of 10.4% **(**Romaine S.T., Potter J., Khanijau A., et al., "Accuracy of a Modified qSOFA Score for Predicting Critical Care Admission in Febrile Children. Pediatrics" 2020;146(4): e20200782), the study required a sample size of 55 subjects with 95% level of confidence and 8% absolute precision, Using Statulator software **(**http://statulator.com/SampleSize/ss1P.html)

Formula used to calculate the sample size:-

$$\mathbf{n = \frac{z^2\, p*q}{d^2}}$$

Where,

Z= Z statistic at α level of significance

d^2= Absolute error

P= Proportion rate

q= 100-p

A total of 55 patients with acute febrile encephalopathy cases between 1 month and 15 years were included. Clinical assessment according to the parameters in the study was done at the point of arrival, along with blood investigations with suspicion of CNS infection (meningitis, seizure, febrile seizure). Peripheral venous blood samples were collected in plain and EDTA tubes during admission. The qSOFA and modified qSOFA scores with serum procalcitonin and NRL ratio were measured.

Statistical Analysis

The data was collected and entered into the predesigned Excel spreadsheet. Statistical analysis was performed by the SPSS program for Windows, version 20.0. Continuous variables were presented as mean ± SD, and categorical variables were presented as absolute numbers and percentages. Results were presented as mean ± SD, median and interquartile range, frequency, percentages, and diagrams. Normally distributed continuous variables between two groups were compared using the independent t-test. For normally distributed variables, the Mann Whitney U test was used. $p<0.05$ was considered statistically significant. Two-tailed tests were performed for all statistical tests. The study scheme is depicted in Figure 1.

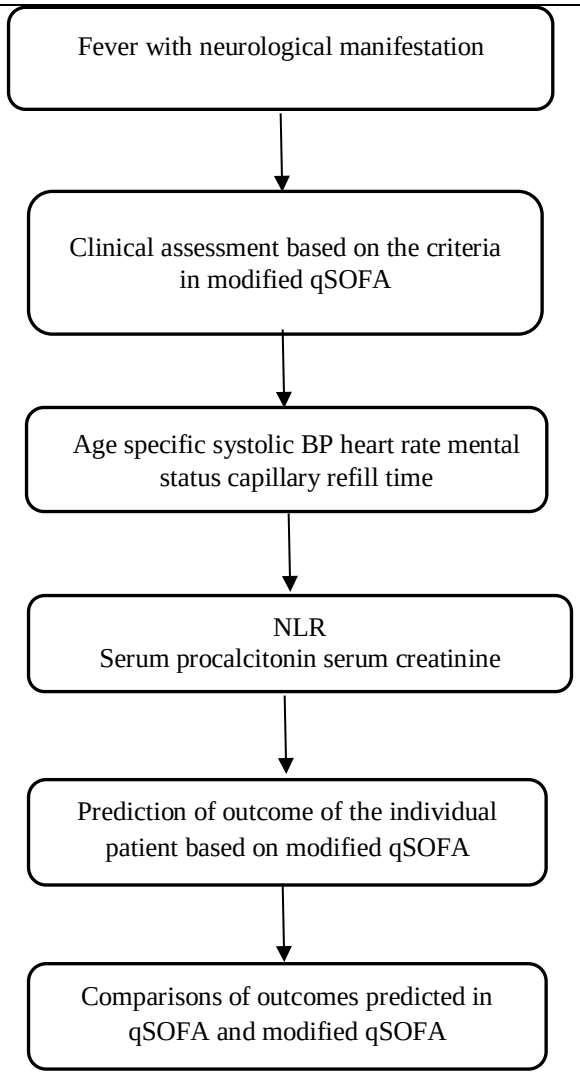


Figure 1: Flow Diagram

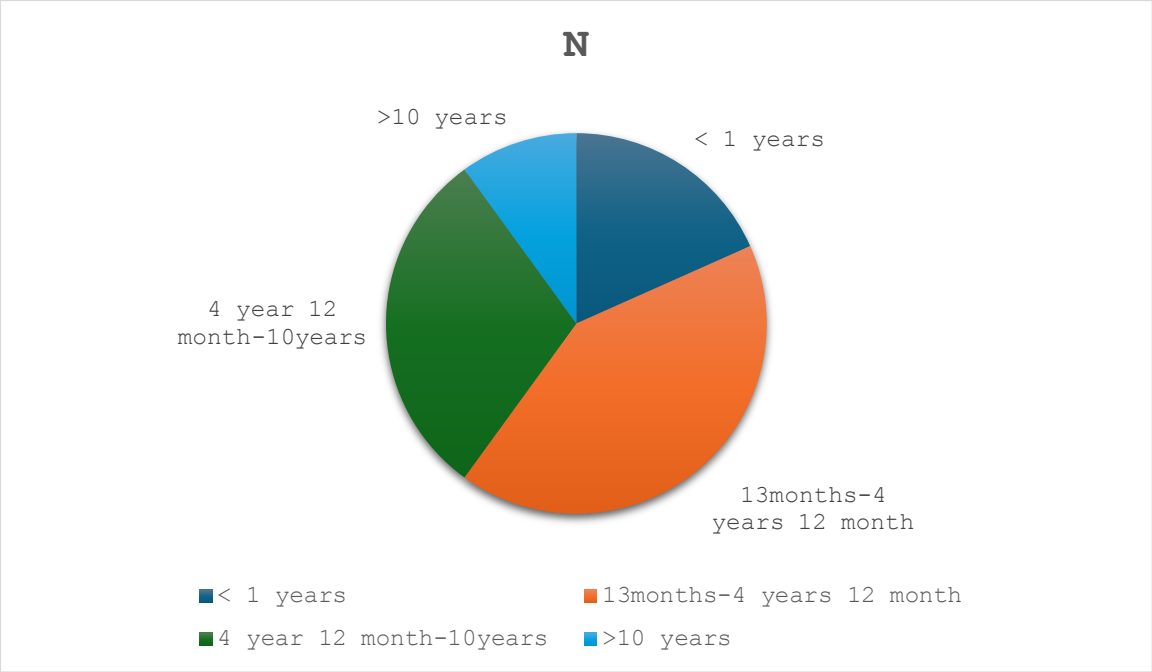
The present study included both qualitative and quantitative variables. Qualitative variables were represented by numbers (%) while quantitative variables were represented by mean ± SD and median (QR). The normality of the data was assessed using the Kolmogorov-Smirnov test. Parametric independent t-tests and non-parametric Mann-Whitney U tests were applied to compare the two quantitative variables. The Fisher exact test was used to determine the association between two independent qualitative variables. The ROC analysis was employed to determine the cut-off value and assess the accuracy of the qSOFA score and modified qSOFA in predicting patient outcomes. A 95% confidence level was considered for all tests. The data analysis was performed using SPSS 20.

RESULTS

Age Group	N	%
< 1 years	11	18.3
13months-4 years 12 month	25	41.7
4 year 12 month-10years	18	30
>10 years	6	10
Total	60	100

Table 1: Age-Wise Distribution

Table 1 depicts the age-wise distribution of the total patients. Out of the total number of patients, the highest were in the age group of 1 to 5 years, while the lowest were above 10 years.

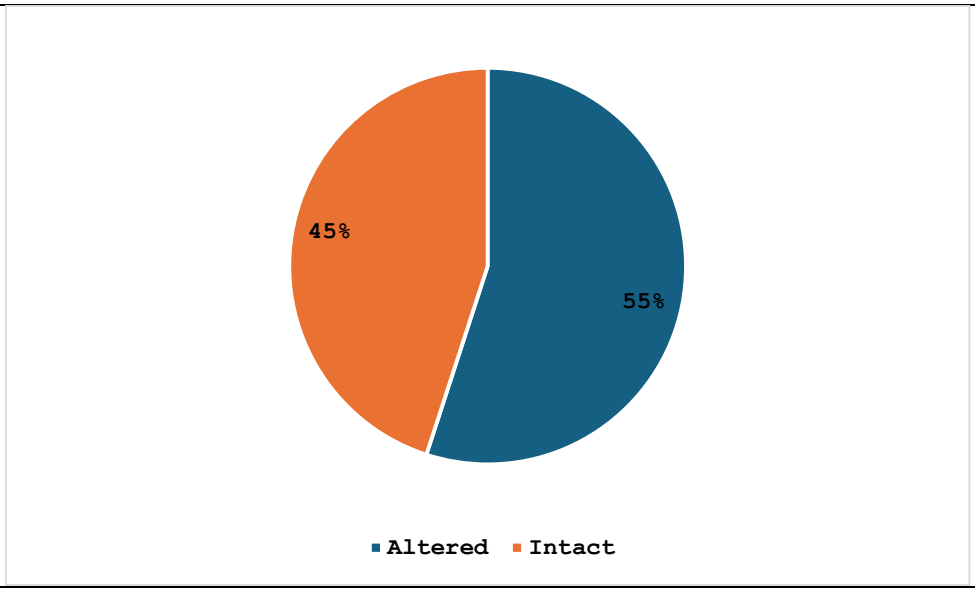


Graph 1: Age-Wise Distribution

Mental Status	Frequency	Percent
Altered	33	55.0
Intact	27	45.0
Total	60	100.0

Table 2: Details of Mental Status

Table 2 depicts the details of the mental status of the total patients. Out of the total number of patients, 55% were altered and 45% intact.

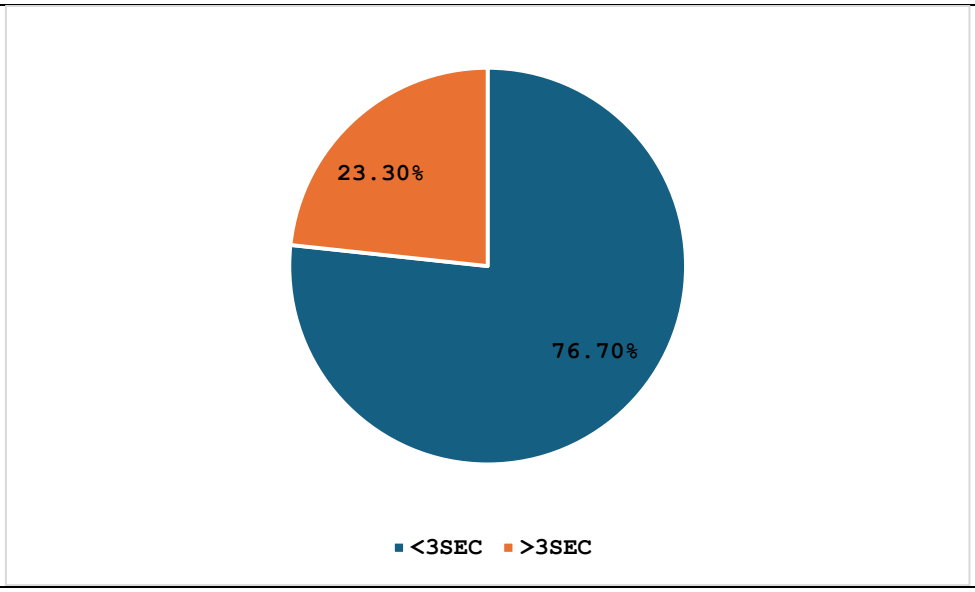


Graph 2: Details of Mental Status

Capillary Refill Time	Frequency	Percent
<3 Seconds	46	76.7
>3 Seconds	14	23.3
Total	60	100.0

Table 3: Details of Capillary Refill Time classification

Table 3 shows the details of the capillary refill time of the total patients. Out of the total number of patients, the highest was 76.7% <3 SEC, while 23.3% had >3 SEC.

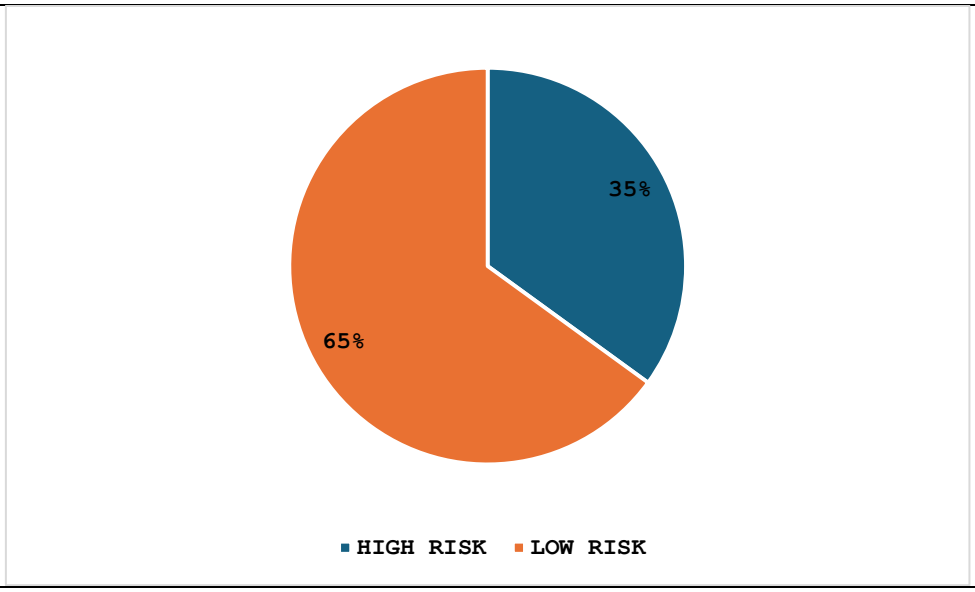


Graph 3: Details of Capillary Refill Time classification

Prognosis Modified qSOFA	Frequency	Percent
High Risk	21	35.0
Low Risk	39	65.0
Total	60	100.0

Table 4: Details of Prognosis Modification qSOFA

Table 4 shows the details of the prognosis of modified qSOFA for total patients. Out of the total number of patients, high risk was observed in 35% and low risk was observed in 65%.

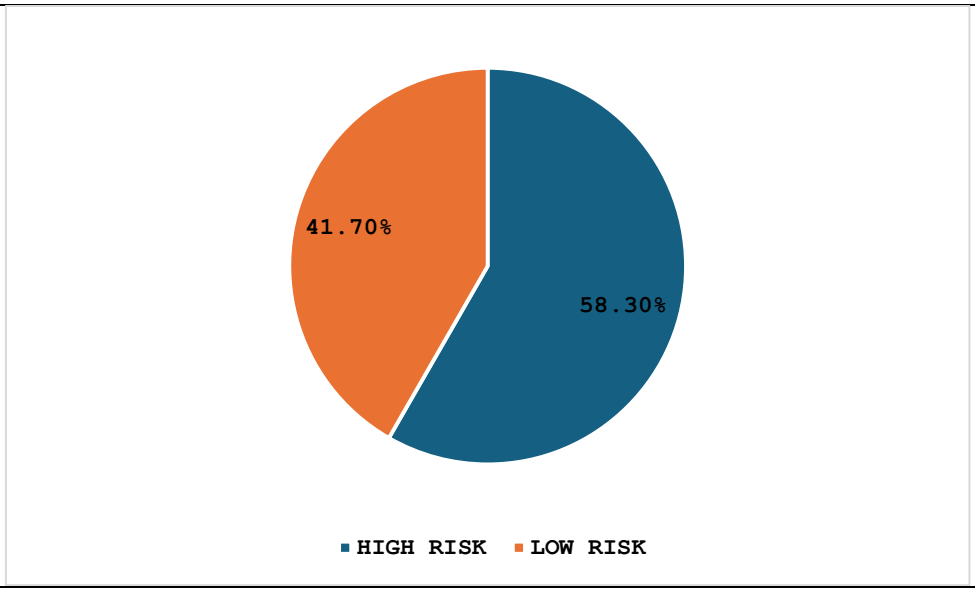


Graph 4: Details of Prognosis Modification qSOFA

Prognosis According to qSOFA	Frequency	Percent
High Risk	35	58.3
Low Risk	25	41.7
Total	60	100.0

Table 5: Details of Prognosis According to qSOFA

Table 5 shows the details of the prognosis for qSOFA in total patients. Out of the total number of patients, high risk was observed in 58.3% and low risk was observed in 41.7%.

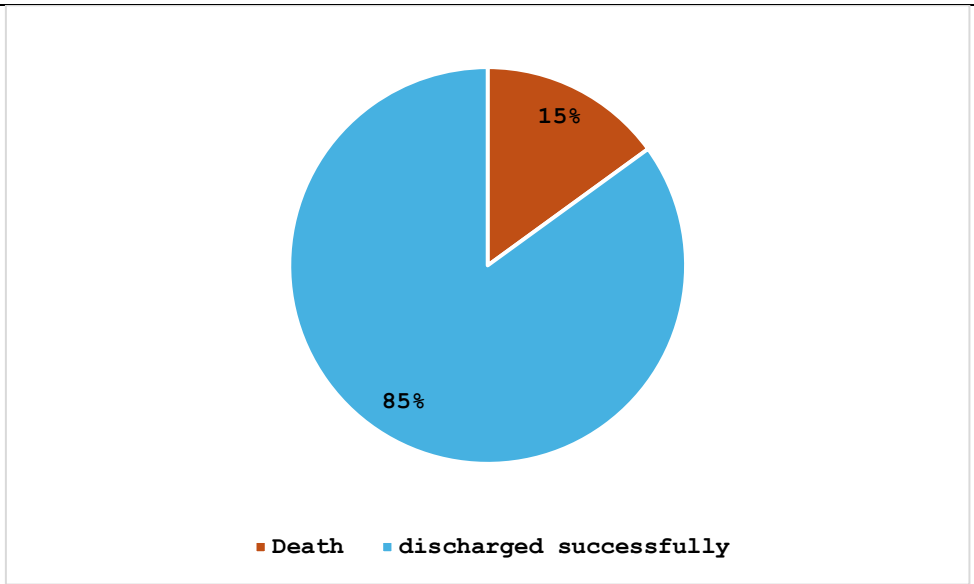


Graph 5: Details of Prognosis According to qSOFA

Outcome	Frequency	Percent
Death	9	15.0
Discharged Successfully	51	85.0
Total	60	100.0

Table 6: Details of the Outcome of the Patients

Table 6 depicts the details of the outcomes for the patients. 85% were discharged successfully, and 15% died.



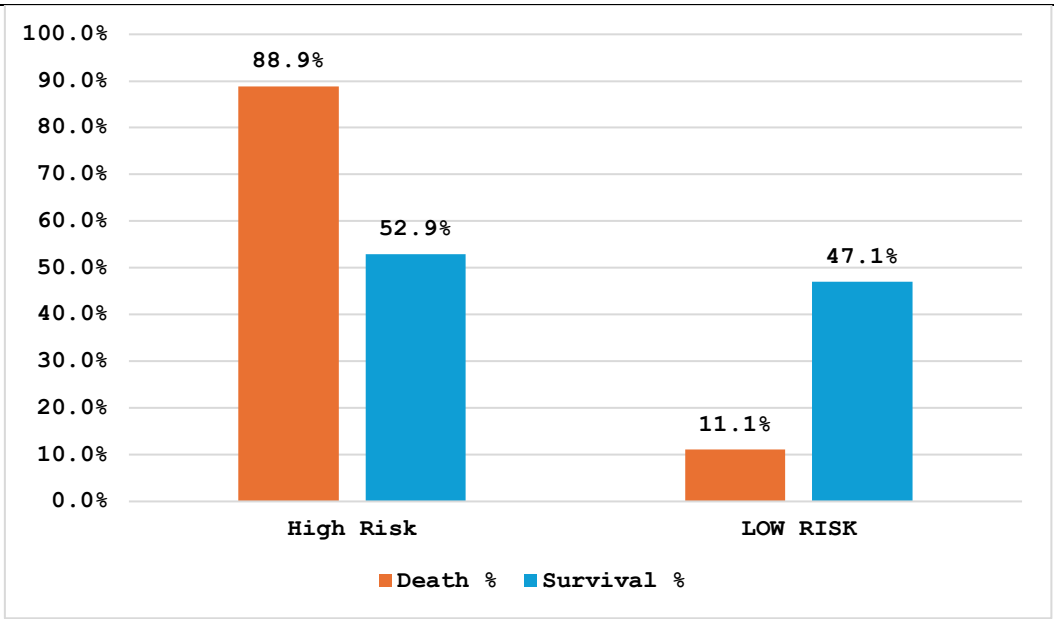
Graph 6: Details of the Outcome of the Patients

Progression According to QSOFA	Outcome				Total
	Death		Survival		
	N	%	N	%	
High Risk	8	88.9%	27	52.9%	35
Low Risk	1	11.1%	24	47.1%	25
Total	9	100.0%	51	100.0%	60

Table 7: Details of Association of Progression According to qSOFA and Outcome of Patients

*Fisher Exact test (P- Value 0.045)

Table 7 shows an association between progression according to qSOFA and the outcome of patients. Out of a total nine patients, one of them passed away and eight (88.9%) had a high risk. The progression, according to the qSOFA, was significantly associated with the outcome of patients. Death was significantly associated with the high risk according to the progression of the qSOFA score. (P < 0.045)



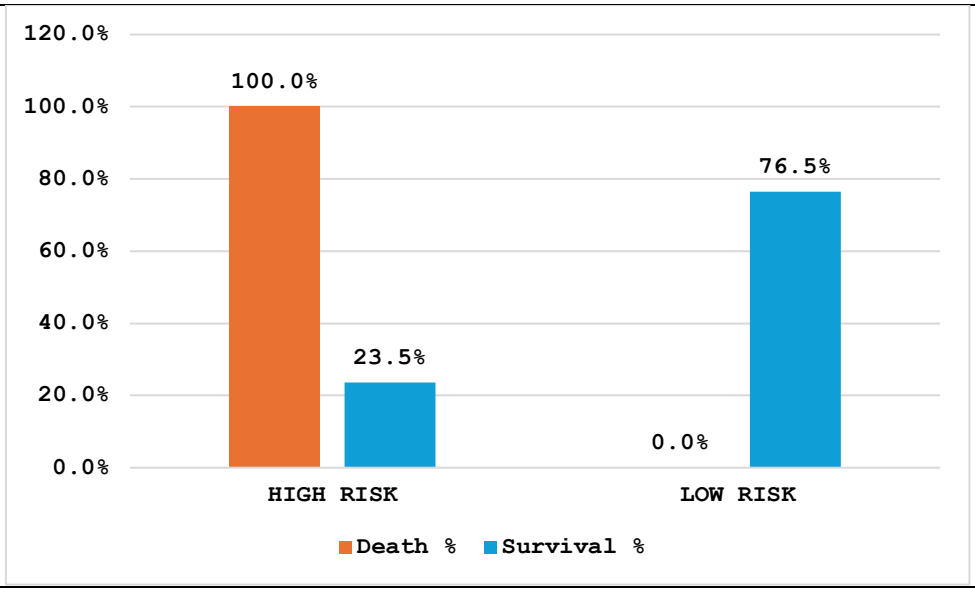
Graph 7: Details of Association of Progression According to qSOFA and Outcome of Patients

Prognosis Modified qSOFA	Outcome				Total
	Death		Survival		
	N	%	N	%	
High Risk	9	100.0%	12	23.5%	21
Low Risk	0	0.0%	39	76.5%	39
Total	9	100.0%	51	100.0%	60

Table 8: Details of Association of Progression According to Modified qSOFA and Outcome of Patients

*Fisher Exact test (P- Value <0.001)

Table 8 shows an association between progression according to modified qSOFA and the outcome of patients. Out of the total of nine patients, none of them passed away, and all nine (100%) had a high risk. The progression according to the modified qSOFA was significantly associated with the outcome of patients. Death was significantly associated with the high risk according to the progression of the modified qSOFA score. (P-value <0.001).



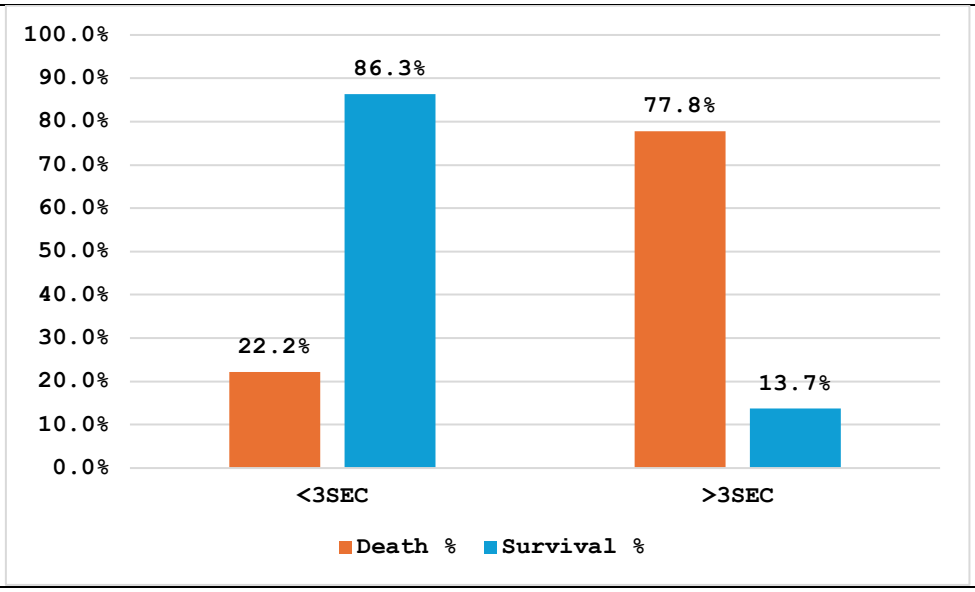
Graph 8: Details of Association of Progression According to Modified qSOFA and Outcome of Patients

Capillary Refill Time	Outcome				Total
	Death		Survival		
	N	%	N	%	
<3 sec	2	22.2%	44	86.3%	46
>3 sec	7	77.8%	7	13.7%	14
Total	9	100.0%	51	100.0%	60

Table 9: Details of Association of Capillary Refill Time and Outcome of Patients

*Fisher Exact test (P-value <0.001)

Table 9 displays the association between capillary refill time and patient outcomes. Out of the total, 9 patients were deceased, with 7 of them (77.8%) exhibiting a capillary refill time of >3 seconds. Conversely, the highest number of surviving patients were observed with a capillary refill time of <3 seconds (86.3%). Death was significantly associated with capillary refill time (>3 seconds). (P-value < 0.001).

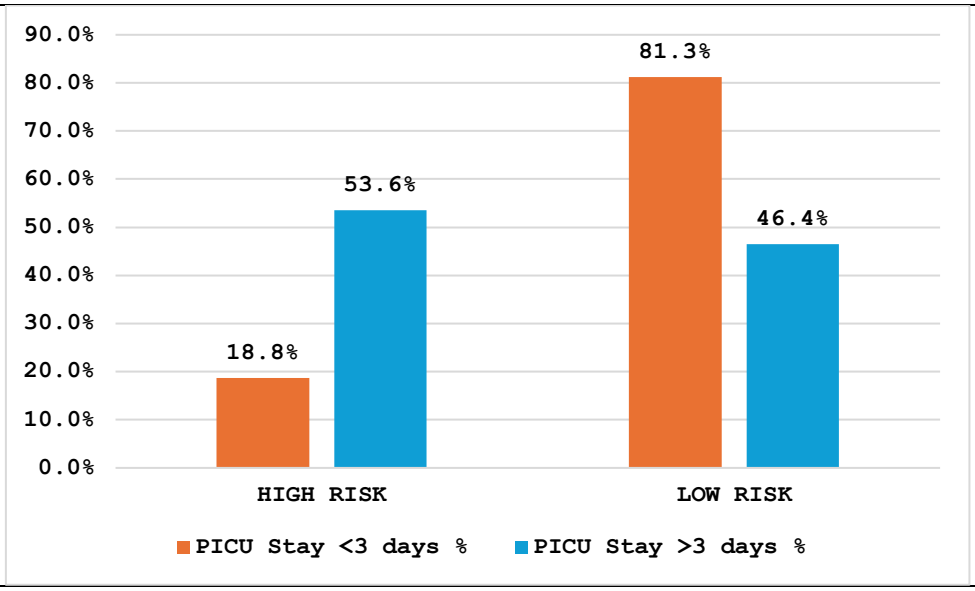


Graph 9: Details of Association of Capillary Refill Time and Outcome of Patients

Prognosis Modified qSOFA	PICU Stay				Total
	<3 Days		>3 Days		
	N	%	N	%	
High Risk	6	18.8%	15	53.6%	21
Low Risk	26	81.3%	13	46.4%	39
Grand Total	**32**	**100.0%**	**28**	**100.0%**	**60**

Table 10: Association between Prognostic Modification and PICU Stay

Table 10 displays the association between the modified qSOFA scoring and the PICU stay required by the patients. 18.8% of patients belonging to high-risk groups require less than 3 days of stay, whereas 53.3% require more than 3 days of stay. In the low-risk category, 81.3% of patients required less than 3 days of stay, and 46.6% of patients required more than 3 days of stay.



Graph 10: Association between Prognostic Modification and PICU Stay

Study Variable	Outcome	N	Mean ± SD	P-Value
Age	Death	9	5.00±5.074	0.976
	Discharge	51	5.04±3.352	
SBP	Death	9	88.89±19.003	0.064
	Discharge	51	98.55±13.231	
DBP	Death	9	56.67±16.583	0.231
	Discharge	51	61.57±10.074	
Respiratory Rate	Death	9	52.56±15.322	P<0.0001
	Discharge	51	31.14±10.813	
NLR	Death	9	2.11±1.833	0.786
	Discharge	51	2.29±1.858	
QSOFA Modified Score	Death	9	5.11±0.601	P<0.0001
	Discharge	51	2.63±1.113	
QSOFA Scoring	Death	9	2.44±0.726	0.005
	Discharge	51	1.61±0.802	
PICU Stay	Death	9	7.56±6.167	0.070
	Discharge	51	4.39±4.464	
Serum Prolcalcitonin	Death	9	19(4-60.50) *	P<0.0001$
	Discharge	51	1(0-2)*	

Table 11: Comparison of Demographic, Vitals and Laboratory Parameters with Outcome of the Patients

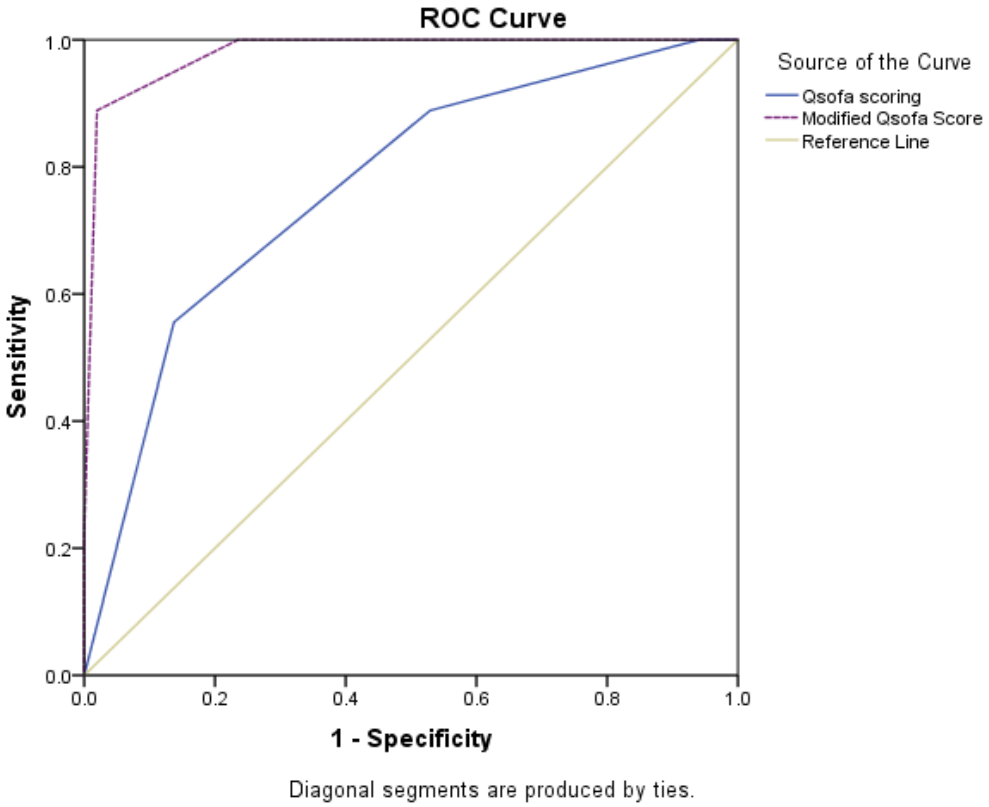
*Median (IQR), $ Mann-Whitney U test applied

Table 11 presents the details of the comparison of demographics, vital signs, and laboratory parameters with patient outcomes. No significant differences were observed in the mean age, SBP, DBP, NLP, or PICU stay between deceased and discharged patients. However, a higher mean respiratory rate (RR), modified qSOFA score, and qSOFA score were observed in deceased patients ($p < 0.001$). Additionally, the median serum prolcalcitonin level was also observed to be higher in deceased patients ($p < 0.0001$).

Test Result Variable(s)	AUC	P- Value	Asymptotic 95% Confidence Interval		Cut-off Value	Sensitivity	Specificity
			Lower Bound	Upper Bound			
qSOFA Score	0.769	0.011	0.603	0.935	≥2.50	55.6%	88.27%
Modified qSOFA Score	0.979	<0.001	0.943	1.000	≥4.50	88.9%	98%

Table 12: Assess the Accuracy of Modified qSOFA Score and qSOFA Score for Predicting Outcomes in Acute Febrile Encephalopathy Patients

Table 12 presents the details of the cut-off values for the qSOFA score and modified qSOFA score to predict outcomes for patients with acute febrile encephalopathy. ROC analysis was conducted on the qSOFA score and modified qSOFA score to determine the most appropriate cut-offs for predicting outcomes for acute febrile encephalopathy patients (Graph 11). Optimal results were observed with a qSOFA score cut-off value of ≥2.50 and a modified qSOFA score cut-off value of ≥4.50 for predicting mortality.



Graph 11: AUC for Diagnostic Accuracy of Modified qSOFA Score and qSOFA Score for Predicting Outcomes in Acute Febrile Encephalopathy Patients

DISCUSSION

In the present study, patients' demographics, vital signs, blood pressure, neutrophil-to-lymphocyte ratio, and serum procalcitonin were noted for the patient's outcome. There was no significant difference in mean age, NLR, or PICU duration between deceased patients and discharged patients. However, the qSOFA score and modified qSOFA were high in the deceased patients ($p < 0.001$). Furthermore, a higher median serum procalcitonin level was also noted in deceased patients ($p < 0.0001$). Death was significantly associated with capillary refill time (>3 seconds).

When used alone or in conjunction with NLR, PCT levels and the modified qSOFA score performed well as predictors of organ failure and death in this investigation. Following the proper stimulation, the serum concentration of procalcitonin increases 1000–10,000 times in 4–12 hours, reaching its peak in 24 hours.[17] Early measurement of the PCT serum level within the first 24 hours after admission is thought to be a good indicator of the severity of the inflammatory process in sepsis, which can ultimately result in organ dysfunction.

A 2017 study on pediatric burn patients reveals that in terms of predicting 30-day mortality, PCT level has 100% sensitivity (95% CI 67.6 to 100%); 15% sensitivity (95% CI: 7.1 to 29.1%); 19% PPV (Positive Predictive Value) (95% CI 10 to 33.3%); and 100% negative predictive value (NPV) (95% CI 61 to 100%).[18]

In 2014, different research found a significant difference (P = 0.0016) between the PCT level of patients with organ failure (PELOD ≥12) and those without (AUC 0.675; P = 0.035). The PCT level cut-off point employed in this investigation was 4.05 ng/mL.[19] According to a research, patients who did not survive had higher NLR levels (P = 0.03), suggesting that NLR level is a good predictor of mortality. Another study produced a contradictory finding, noting that there was no relationship between the NLR level and the 28-day death rate in individuals with sepsis. Adult patients were the topic of all the aforementioned studies.[20-22]

We advise against using NLR alone as a predictor of organ failure and/or death in pediatric sepsis since high neutrophil counts and/or lymphopenia can be brought on by a number of illnesses other than infection. It is important to remember that NLR has a strong sensitivity to predict organ dysfunction, making it a useful screening tool for individuals who require further septic work-up or surveillance of organ failure. Our investigation revealed that the cutoff values for the qSOFA and modified qSOFA are ≥2.5 and ≥4.50, respectively. The AUCs (0.769 vs. 0.979) of this study indicate that these levels can function as better indicators of organ dysfunction than the qSOFA, but the modified qSOFA score can be a more accurate predictor than the qSOFA, exhibiting high sensitivity of 88.9% and specificity of 98% with a p-value of 0.001. Whereas a confirmation tool needs high specificity, a screening tool needs great sensitivity. Like earlier external validation studies, ours also verified that the modified qSOFA score and the qSOFA score had good

specificity but low sensitivity.[23] When all three of these parameters—modified qSOFA, PCT, and NLR-are combined, they also demonstrate high performance as predictors of organ dysfunction.

According to Romaine et al.'s study, a large independent validation cohort of febrile ED patients indicated that the LqSOFA score, a novel, rapid, bedside score for children, had strong discriminative power in predicting CC admission.[24] Finding the "needle in the haystack"-children in need of immediate care and more investigation—is crucial in the emergency department. Given the low frequency of sepsis, identifying children who may need CC hospitalization may be a more valuable use of a sepsis-specific score in a pediatric emergency department.

For two reasons, we want to keep using the updated qSOFA as a validation mechanism. First, it is confirmed to be a straightforward instrument with excellent specificity (88.27% and 98%) and high specificity. Second, obtaining the full SOFA score could be difficult in clinical settings other than the ICU, such as the ED. In summary, we think the newly presented algorithm is the best clinical decision rule in clinical settings outside of the ICU and the most optimal use of biomarker information, employing high-sensitivity modified qSOFA as a screening tool and a confirmation tool.

This study's inability to account for every confounding factor that affects organ malfunction and death was one of its limitations. The actual treatment of sepsis was one of the primary confounding variables. In

order to reduce confounding variables that might affect organ dysfunction and the mortality of pediatric sepsis patients, we recommend conducting cohort research using the proper sepsis care procedures.

SUMMARY

A hospital-based, prospective observational study was conducted in the PICU, Shri BM Patil Medical College Hospital and Research Centre, Vijayapura, to assess the accuracy of modified qSOFA with serum procalcitonin and neutrophil-leucocyte ratio for predicting outcome in acute febrile encephalopathy in children. The study included 60 patients with suspected central nervous system (CNS) infections admitted to the PICU who fulfilled the inclusion criteria. The study was conducted for a period of one and a half years, from June 2022 to January 2024. Among 60 patients, the majority fell within the age range of 1-5 years, with the fewest being above 10 years old. Out of the 60 patients, 55% underwent alterations while 45% remained intact. The majority of patients, 76.7%, had capillary refill time less than 3 seconds, whereas 23.3% had more than 3 seconds of capillary refill time. High-risk patients accounted for 35% of the total, while low-risk patients made up 65%. Among 60 (100%) patients, 85% of the patients were discharged while 15% of the patients died. Among the 9 patients who died, about 8 (88.9%) were classified as high risk. The progression based on the modified QSOFA was found to have a significant association with the patients' outcomes. The occurrence of death was significantly linked to high risk based on the progression of the modified QSOFA score ($P<0.045$). In the high-risk group, 18.8% of patients required a PICU stay of less than 3 days, while

53.3% needed more than 3 days of PICU stay. For the low-risk category, 81.3% of patients had a PICU stay of less than 3 days, whereas 46.6% required more than 3 days of PICU stay.To predict outcomes for patients with acute febrile encephalopathy, a ROC analysis was performed on the qSOFA score and modified qSOFA score. The optimal cut-off value for the qSOFA score was ≥2.50, while for the modified qSOFA score, it was ≥4.50, both indicating a higher likelihood of mortality.

CONCLUSION

- The highest number of patients belonged to age-group 2 to 5 years followed by 5-10years

- The progression according to the qSOFA and modified qSOFA and the outcome of the patients were significantly correlated with the outcome of the patients.
- The serum prolcalcitonin level was higher in deceased patients.
- Modified qSOFA has high sensitivity and high specificity in predicting mortality due to acute febrile encephalopathy . Whereas the qSOFA has low sensitivity and high specificity in predicting mortality due to acute febrile encephalopathy.
- It was demonstrated the superior performance of modified qSOFA over qSOFA and PCT to identify patients with a high risk of mortality and a poor prognosis.
- With our study, we provide further evidence that the modified qSOFA has the prognostic ability to justify its introduction into routine practice in the pediatric ED.
- The incorporation of the ordinal scale of PCT into the modified qSOFA model could enhance sensitivity and reclassify patients into risk groups that better reflect their actual short-term mortality risk.
- The modified qSOFA requires further evaluation in other settings before recommending more widespread use as an entry criterion into randomized controlled trials in the pediatric emergency department.

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RESEARCH INFORMED CONSENT FORM

BLDE(DU) Shri B.M. PATIL Medical College, Hospital & Research Centre,Vijayapura-586103.

TITLE OF THE TOPIC: **MODIFIED QSOFA SCORE WITH SERUM PROCALCITONIN AND NEUTROPHIL AND LYMPHOCYTE RATIO FOR PREDICTING THE OUTCOME FOR ACUTE FEBRILE ENCEPHALOPATHY IN CHILDREN**

GUIDE: **DR.S V PATIL MD**
PROFESSOR
DEPARTMENT OF PEDIATRICS

PG STUDENT **DR.ANWITA SINHA**
POSTGRADUATE
DEPARTMENT OF PEDIATRICS

ANNEXURE I

ETHICAL CLEARANCE CERTIFICATE OBTAINED BY THE INSTITUTION

BLDE
(DEEMED TO BE UNIVERSITY)
Declared as Deemed to be University u/s 3 of UGC Act, 1956
Accredited with 'A' Grade by NAAC (Cycle-2)
The Constituent College
SHRI B. M. PATIL MEDICAL COLLEGE, HOSPITAL & RESEARCH CENTRE, VIJAYAPURA

Azadi Ka Amrit Mahotsav

BLDE (DU)/IEC/ 649/2022-23

30/8/2022

INSTITUTIONAL ETHICAL CLEARANCE CERTIFICATE

The Ethical Committee of this University met on **Friday, 26th August, 2022 at 3.30 p.m. in the Department of Pharmacology** scrutinizes the Synopsis of Post Graduate Student of BLDE (DU)'s Shri B.M.Patil Medical College Hospital & Research Centre 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: "MODIFIED QSOFA SCORE WITH SERUM PROCALCTONIN AND NEUTROPHIL AND LEUCOCYTE RATIO FOR PREDICTING THE OUTCOME FOR ACUTE FEBRILE ENCEPHALOPATHY IN CHILDREN".

NAME OF THE STUDENT/PRINCIPAL INVESTIGATOR: DR. ANWITA SINHA

NAME OF THE GUIDE: DR S V PATIL, Professor, Dept. of Pediatrics.

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

Chairman,
utional Ethical Committee,
(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

llowing documents were placed before Ethical Committee for Scrutinization.

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

angaramma Sajjan Campus, B. M. Patil Road (Sholapur Road), Vijayapura - 586103, Karnataka, India.
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College: Phone: +918352-262770, Fax: +918352-263019, E-mail: bmpmc.principal@bldedu.ac.in

PURPOSE OF RESEARCH:

I have been told that the present study will help in assessing the "**MODIFIED QSOFA SCORE WITH SERUM PROCALCITONIN AND NEUTROPHIL AND LYMPHOCYTE RATIO FOR PREDICTING THE OUTCOME FOR ACUTE FEBRILE ENCEPHALOPATHY IN CHILDREN**

PROCEDURE:

I do understand that after having obtained a detailed clinical history, thorough clinical examination and relevant investigations, a prospective study **MODIFIED QSOFA SCORE WITH SERUM PROCALCITONIN AND NEUROPHIL AND LYMPHOCYTE RATIO FOR PREDICTING THE OUTCOME FOR ACUTE FEBRILE ENCEPHALOPATHY IN CHILDREN**

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 ANWITA SINHA 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 Anwita Sinha 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. I 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. ANWITA SINHA Date

(Investigator)

PARENTS / GUARDIAN CONSENT STATEMENT:

We confirm that Dr ANWITA SINHA is doing a study "**MODIFIED QSOFA SCORE WITH SERUM PROCALCITONIN AND NEUTROPHIL AND LYMPHOCYTE RATIO FOR PREDICTING THE OUTCOME FOR ACUTE FEBRILE ENCEPHALOPATHY IN CHILDREN**"

A prospective study. Dr ANWITA SINHA 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) Date

SCHEME OF CASE TAKING

Name :

Age :

Sex :

IP NO :

Chief complaint :

Past history: significant / not significant, if significant specify

Birth history: significant / not significant, if significant specify

Antenatal history

Natal history

Postnatal history

Family history :

PARAMETERS	SCORE
SYSTOLIC BLOOD PRESSURE	
RESPIRATORY RATE	
MENTAL STATUS	
CAPILLARY REFIL TIME	
SERUM PROCALCITONIN	
NLR RATIO	
TOTAL QSOFA MODIFIED QSOFA	

Prognosis

MODIFIED qSOFA	qSOFA
HIGH RISK / LOW RISK	HIGH RISK / LOW RISK

OUTCOME:

PICU STAY :

	0	1
SYSTOLIC BLOOD PRESSURE	TERM-1 MONTH -<60 2-12MONTH-<70 1-4YEARS-<75 5-12-<80 >12- <90	TERM-1 MONTH ->60 2-12MONTH->70 1-4YEARS->75 5-12->80 >12- >90
RESPIRATORY RATE	<1YR →30-55 1-2YR →20-30 3-5YR →20-25 6-11YR → 14-22 12-15YR →12-18	>55 >30 >25 >22 >18
MENTAL STATUS	NORMAL MENTAL STATUS	ALTERED MENTAL STATUS
CAPILLARY REFIL TIME	<3SECS	>3SECS
SERUM PROCALCITONIN	<2	>2
NLR RATIO	<1.13	>1.13

age	Sex	IP Numb	Chief Complaint	past hist	Birth History	antenatal history	Natal history	Postnatal history	family history	Blood press	Respirat	Mental S	Capillary	Serum P	NLR	SCORE	prognos	qsofa sc	prognos	PICU St	outcome
8 years	Female	954714	c/o fever since 5days , altered sensorium since 1	Not signif	Not Significant	BOOKED CASE	NVD/CRIED AT BIRTH/ B.\	NO H/O NICU ADMISSION	non consanginious	100/60MMHG	26/MIN	altered	<3SEC	0.71	5.3	3	LOW RIS	2	HIGH RIS	4 DAYS	discharged sucessfully
13YEARS	Male	18245	C/O FEVER SINCE 2 DAYS, C/O CONVULSION	Significar	Not Significant	BOOKED CASE	TERM/NVD/CRIED AT BIR	NO H/O NICU ADMISSION	non consanginious	120/70MMHG	42/MIN	altered	>3SEC	6.01	1.2	6	HIGH RIS	3	HIGH RIS	2 DAYS	Death
1 YEAR	Female	193934	C/O FEVER SINCE 4 DAYS , C/O CONVULSION	Not signif	Significant	BOOKED CASE	NVD/DIDNOT CRY AT BIR	H/O ADMISSION FOR RDS F	consanginious	60/40MMHG `	48/MIN	altered	>3SEC	5.93	1.4	5	HIGH RIS	2	HIGH RIS	7 DAYS	Death
5 YEARS	Male	127321	C/O FEVER SINCE 2 DAYS ALTERED SENSOF	Not signif	Not Significant	BOOKED CASE	NVD/CIVIL HOSPITAL/CRI	NO H/O NICU ADMISSION	non consanginious	90/40 mmhg	32/min	altered	<3SEC	1.16	0.76	3	LOW RIS	3	HIGH RIS	3 days	discharged sucessfully
5 months	Female	193803	c/o fever since 1 day c/o convulsion 3 episode	Not signif	Not Significant	BOOKED CASE	term/lscs/cried at birth/	NO H/O NICU ADMISSION	consanginious	70/40 MM HG	36/MIN	INTACT	<3SEC	0.063	0.2	0	LOW RIS	0	LOW RIS	2 DAYS	discharged sucessfully
3 YEARS	Male	192627	C/O FEVER SINCE 1 WEEK, C/O CONVULSION	Significar	Not Significant	BOOKED CASE	TERM/LSCS/CRIED AT BII	NO H/O NICU ADMISSION	consanginious	80/50MMHG	28/MIN	intact	>3SEC	1.15	0.3	2	LOW RIS	1	LOW RIS	3 DAYS	discharged sucessfully
13 YEARS	Male	171031	C/O FEVER SINCE 2 DAYS AND 1 EPISODE WI	Not signif	Not Significant	BOOKED CASE	TERM/LSCS/CRIED AT BII	NO H/O NICU ADMISSION	consanginious	120/70	23/MIN	intact	<3SEC	2.9	5.13	4	HIGH RIS	2	HIGH RIS	5 DAYS	DAMA
4 YEARS	Female	165901	C/O FVER SINCE 4 DAYS, C/O CONVULSION 2	Not signif	Not Significant	BOOKED CASE	TERM/NVD/B.WT-2.5	NO H/O NICU ADMISSION	non consanginious	100/70MMHG	22/MIN	intact	<3SEC	1.2	3.6	2	LOW RIS	1	LOW RIS	4 DAYS	discharged sucessfully
3 YEARS	Male	164226	C/O FEVER SINCE 2 DAYS AND CONVULSION	Significar	Significant	BOOKED CASE	TERM/LSCS/CIED AT BIR	NO H/O NICU ADMISSION	non consanginious	90/60MMHG	28/MIN	intact	<3SEC	0.2	1.5	2	LOW RIS	1	LOW RIS	2 DAYS	discharged sucessfully
5 YEARS	Male	158393	C/O FEVER SINCE 2 DAYS AND 2 EOISODE OF	Not signif	Not Significant	BOOKED CASE	TERM/NVD/CRIED IMMED	H/O NICU ADMISSION FOR	consanginious	110/70 MMHG	24/MIN	intact	<3SEC	0.3	4.8	3	LOW RIS	2	HIGH RIS	2 DAYS	discharged sucessfully
13YEARS	Male	2310	C/O FEVER SINCE 3 DAYS AND ALTERED SEI	Not signif	Not Significant	BOOKED CASE	TERM/NVD/CRIED AT BIR	NO H/O NICU ADMISSION	consanginious	100/80MMHG	19/MIN	intact	<3SEC	0.18	1.14	2	LOW RIS	1	LOW RIS	2 DAYS	discharged sucessfully
1 YEAR	Female	145960	C/O FEVER SINCE 4 DAYS COUGH AND COLD	Not signif	Not Significant	BOOKED CASE	TERM/LSCS/CRIED AT BII	NO H/O NICU ADMISSION	non consanginious	90/70MMHG	82/MIN	ALTERED	>3SEC	21.5	5.33	6	HIGH RIS	3	HIGH RIS	21 DAYS	Death
12 YEARS	Female	109818	C/O FEVER SINCE 1 DAY, C/O CONVULSION 4 I	Significar	Significant	BOOKED CASE	TERM/NVD/DIDNOT CRY /	H/O NICU ADMISSION FOR	non consanginious	110/60 MM HG	16/MIN	altered	<3SEC	1.1	0.7	1	LOW RIS	1	LOW RIS	3 DAYS	discharged sucessfully
3 YEARS	Male	54301	C/O 1 EPISODE OF CONVULSION 1 DAY BACK	Not signif	Not Significant	BOOKED CASE	LATE PRETERM/AGA/LS(	NO H/O NICU ADMISSION	non consanginious	106/60MMHG	24/MIN	intact	<3SEC	0.388	1.5	2	LOW RIS	1	LOW RIS	3 DAYS	discharged sucessfully
4 YEARS	Male	11564	C/O FEVER SINCE 5 DAYS , C/O SEZIURE EPIS	Significar	Significant	BOOKED CASE	TERM/NVD/CRIED AT BIR	H/O NICU ADMISSION FOR	consanginious	100/70MMG HG	40CPM	altered	<3SEC	20	4.3	5	HIGH RIS	3	HIGH RIS	6 DAYS	Death
2YEARS	Male	156103	C/O FVER SINCE 2 DAYS , C/O	Not signif	Significant	BOOKED CASE	NVD/DIDNOT CRY AT BIR	H/O NICU ADMISSION FOR	non consanginious	100/70	30/MIN	altered	<3SEC	0.051	2.4	4	HIGH RIS	3	HIGH RIS	3 DAYS	discharged sucessfully
2 YAERS	Female	311532	C/O COUGH SINCE 2 DAYS AND C/O FEVER SI	Not signif	Not Significant	BOOKED CASE	LATE PRETERM/AGA/NVD/(	NO H/O NICU ADMISSION	consanginious	100/60MMHG	28/MIN	intact	<3SEC	0.054	5	3	LOW RIS	2	HIGH RIS	3 DAYS	discharged sucessfully
4 YEARS	Male	312090	C/O FEVER SINCE 3 DAYS, COUGH SINCE 3 D.	Significar	Significant	BOOKED CASE	LATE PRETERM/AGA/DID!	H/O NICU ADMISSION FOR	non consanginious	90/60MMHG	48/MIN	altered	<3SEC	0.365	6.4	3	LOW RIS	2	HIGH RIS	3 DAYS	discharged sucessfully
10 YEARS	Female	31709	C/O FEVER SINCE 1 DAY C/O SEZIURE 1 EPISC	Not signif	Not Significant	BOOKED CASE	TERM/NVD/CRIED AT BIR	NO H/O NICU ADMISSION	consanginious	130/90MMHG	40/MIN	intact	<3SEC	0.15	1	2	LOW RIS	2	HIGH RIS	2 DAYS	discharged sucessfully
3 YEARS	Female	917726	C/O FEVER SINCE 2 DAYS, ALTERED SENSOF	Not signif	Not Significant	BOOKED CASE	NVD/CRIED AT BIRTH	NO H/O NICU ADMISSION	non consanginious	80/50MMHG	20/MIN	altered	<3SEC	0.06	1.02	1	LOW RIS	1	LOW RIS	2 DAYS	discharged sucessfully
5 YEARS	Male	91482	C/O FEVER AND IRRATIBILITY SINCE TWO D/	Not signif	Not Significant	BOOKED CASE	TERM/LSCS/CRIED AT BII	NO H/O NICU ADMISSION	non consanginious	90/60MMHG	18/MIN	intact	<3SEC	0.1	1.06	0	LOW RIS	1	LOW RIS	1 DAYS	discharged sucessfully
4 YEARS	Male	317716	C/O FEVER SINCE 5 DAYS , 2 EPISODE OF CC	Significar	Not Significant	BOOKED CASE	TERM/LSCS/CRIED AT BII	NO H/O NICU ADMISSION	non consanginious	80/60 MM HG	24/MIN	intact	<3SEC	0.66	2.1	3	LOW RIS	2	HIGH RIS	3 DAYS	discharged sucessfully
2 1/2 YEAI	Male	348333	C/O FEVER SINCE 1 DAY CONVULSION 1 EPISI	Not signif	Not Significant	BOOKED CASE	LATE PRETERM/NVD/CRIE	NO H/O NICU ADMISSION	consanginious	80/50MMHG	30/MIN	intact	>3SEC	0.2	10.2	3	LOW RIS	1	LOW RIS	4 DAYS	discharged sucessfully
2 YAER 8	Male	156103	C/O SEZIURE, C/O FEVER SINCE 1 DAY	Significar	Not Significant	BOOKED CASE	TERM/NVD/CIAB	H/O NICU ADMISSION FOR	non consanginious	90/60MMHG	30/MIN	altered	>3SEC	0.051	2.4	3	LOW RIS	1	LOW RIS	5 DAYS	discharged sucessfully
12 YEARS	Female	298635	C/O FEVER SINCE 3 DAYS AND SEZIURE FOR	Significar	Not Significant	BOOKED CASE	TERM/NVD/CRIED AT BIR	NO H/O NICU ADMISSION	non consanginious	90/60MMHG	30/MIN	altered	>3SEC	2.246	2	5	HIGH RIS	2	HIGH RIS	6 DAYS	Death
1 1/2 YEAI	Male	6061	C/O FEVER SINCE 1 DAY AND MULTIPLE CON	Significar	Not Significant	BOOKED CASE	NO H/O NICU ADMISSION	TERM/NVD/CRIED AT BIRT	consanginious	90/40MMHG	48/MIN	altered	<3SEC	0.9	0.8	2	LOW RIS	2	HIGH RIS	3 DAYS	discharged sucessfully
5 YEARS	Male	732545	C/O FEVER SINCE 1 DAY AND 2 EPISODE OF C	Significar	Significant	BOOKED CASE	TERM/NVD/DIDNOT CRY /	H/O NICU ADMISSION FOR	consanginious	100/70 MMHG	47/MIN	altered	>3SEC	0.297	0.7	2	LOW RIS	3	HIGH RIS	10 DAYS	discharged sucessfully
4MONTH!	Female	584965	C/O COUGH 4DAYS, C/O FEVER SINCE 4 DAYS	Not signif	Not Significant	BOOKED CASE	TERM/LSCS/CRIED AT BII	H/O NICU ADMISSION FOR	consanginious	90/50MMHG	48/MIN	altered	<3SEC	0.722	0.61	2	LOW RIS	3	HIGH RIS	4 DAYS	discharged sucessfully
6MONTH!	Female	155581	C/O FEVER SINCE 3 DAYS, LETHARGY SINCE	Not signif	Not Significant	BOOKED CASE	TERM/LSCS/CRIED AT BII	H/O NICU ADMISSION	consanginious	80/40 MMHG	46/MIN	altered	>3SEC	2	0.4	4	HIGH RIS	2	HIGH RIS	2 DAYS	Death
10 YEARS	Female	137025	C/O FEVER SINCE 2 DAYS AND SEZIURE 2 EP	Significar	Not Significant	BOOKED CASE	TERM/NVD/CRIED AT BIR	NO H/O NICU ADMISSION	consanginious	90/60MMHG	23/MIN	intact	>3SEC	0.163	1.5	3	LOW RIS	1	LOW RIS	3 DAYS	DAMA
6 YEAR	Male	95932	C/O FEVER 6 DAYS, RASHES SINCE 5 DAYS L	Not signif	Not Significant	BOOKED CASE	TERM/NVD/CRIED AT BIR	NO H/O NICU ADMISSION	consanginious	90/50 MMHG	24/MIN	intact	<3SEC	0.8	0.15	2	LOW RIS	2	HIGH RIS	6 DAYS	discharged sucessfully
8 YEARS	Female	94714	C/O FEVER SINCE 5 DAYS AND C/O ALTERED	Not signif	Not Significant	BOOKED CASE	NVD/CRIED AT BIRTH/B.W	NO H/O NICU ADMISSION	non consanginious	120/70MMHG	26CPM	altered	<3SEC	0.71	5.3	4	HIGH RIS	2	HIGH RIS	4 DAYS	DAMA
3 1/2 YEAI	Female	58601	C/O FEVER SINCE 1 DAYS , COUGH SINCE 1 D/	Significar	Significant	BOOKED CASE	NVD/TERM/DIDNOT CRY /	H/O NICU ADMISSION IVIO	non consanginious	80/60MMHG `	36/MIN	intact	>3SEC	1.7	3.8	3	LOW RIS	0	LOW RIS	4 DAYS	discharged sucessfully
3YEARS	Male	54301	C/O FEVER SINCE 4 DAYS AND 1 EPISODE OF	Not signif	Not Significant	BOOKED CASE	TERM/LSCS/ CRIED AT BI	H/O H/O NICU AD,ISSION	non consanginious	110/60MMHG	24/MIN	altered	<3SEC	0.388	1.5	3	LOW RIS	2	HIGH RIS	2 DAYS	discharged sucessfully
9 MONTH	Male	101074	C/O FEVR SINCE 2 DAYS , C/O COLD AND COL	Not signif	Not Significant	BOOKED CASE	TERM/AGA/NVD	NO H/O NICU ADMISSION	non consanginious	70/40MMHG	42/MIN	intact	<3SEC	1.36	1.5	2	LOW RIS	1	LOW RIS	1 DAY	discharged sucessfully
1y 2 month	Male	54110	C/0 fever & 2 episodes of convulsions	Significar	Not Significant	BOOKED CASE	Term/aga/cried at birth	No h/0 nicu admission	non consanginious	100/60	38/min	altered	<3SEC	17.06	2	4	HIGH RIS	3	HIGH RIS	3 days	discharged sucessfully
3years	Male	31167	C/0 fever since 3 days, c/0 cough since 3 days, c/	Not signif	Not Significant	BOOKED CASE	Term/aga/male/cried immec	No h/0 nicu admission	non consanginious	100/70	48/min	altered	<3SEC	2.066	3.1	4	HIGH RIS	1	LOW RIS	24 days	discharged sucessfully
6 years	Female	54111	C/0 cough & cold since 15 days, c/0 2 episodes of	Not signif	Not Significant	BOOKED CASE	Nvd/term/ciab	No h/0 nicu admission	consanginious	110/60	28/min	altered	<3SEC	0.064	0.7	2	LOW RIS	1	LOW RIS	4 days	discharged sucessfully
7 years	Male	13621	C/o fever since 4 days , c/o altered sensorium for	Not signif	Not Significant	BOOKED CASE	Term/aga/male/cried at birt	No h/o nicu admisssion	consanginious	110/70mmhg	24/MIN	intact	<3SEC	0.087	1.02	1	LOW RIS	1	LOW RIS	2 days	discharged sucessfully
8yrs	Male	44543	C/0 fever since 5 days,c/0 cough since 3 days, c/(	Not signif	Not Significant	BOOKED CASE	Term/aga/nvd/ciab	No h/0 nicu admission	non consanginious	110/80	48/min	altered	>3SEC	19.016	1.7	5	HIGH RIS	1	LOW RIS	2 days	Death
11 years	Male	24326	C/o 2 episodes of seizures	Significar	Not Significant	BOOKED CASE	Late preterm/aga/male/crie	No h/o nicu admission	consanginious	100/60mmhg	28cpm	intact	<3SEC	3.6	2.1	3	LOW RIS	1	LOW RIS	4 days	discharged sucessfully
3 years	Male	56322	C/o fever for 2 days, rolling of eyes for 1 days	Significar	Not Significant	BOOKED CASE	Term/aga/male/cried at birt	No h/o nicu admission	non consanginious	90/60mmhg	24/MIN	intact	<3SEC	0.03	1.1	3	LOW RIS	2	HIGH RIS	3 data	discharged sucessfully
7 years	Female	35704	C/0 fever 7 days, c/0 headache since 7 days, c/0 \	Not signif	Not Significant	BOOKED CASE	Term/lscs/cried at birth	H/0 nicu admission on dol-3	non consanginious	120/80	32/min	altered	<3SEC	0.036	2.1	4	HIGH RIS	2	HIGH RIS	6 days	discharged sucessfully
9 months	Female	14952	C/o fever, c/o alerted sensorium, c/o vomiting	Not signif	Not Significant	BOOKED CASE	Pre term/aga/female/ cried a	No h/o nicu admission	non consanginious	120/60mmhg	28 cpm	intact	<3SEC	3.01	4.2	4	HIGH RIS	1	LOW RIS	12days	discharged sucessfully
7 YEARS	Male	13621	C/O FEVER SINCE 4 DAYS AND ALTERED SEI	Not signif	Not Significant	BOOKED CASE	TERM/AGA/CRIED AT BIR	NO H/O NICU ADMISSION	consanginious	100/70MMHG	24/MIN	intact	<3SEC	0.087	1.02	1	LOW RIS	2	HIGH RIS	2 DAYS	discharged sucessfully
6 years	Female	12432	C/o cough, cold since 15 days; c/o 2 episodes of c	Not signif	Not Significant	BOOKED CASE	NVD/cried at birth/b wt 2kg	No h/o nicu admission	consanginious	110/60mmhg	28/min	altered	<3SEC	0.065	0.7	2	LOW RIS	1	LOW RIS	4 days	discharged sucessfully
3 years	Male	31167	C/o fever since 3 days; c/o cough since 3 days; c/	Not signif	Not Significant	BOOKED CASE	LSCS/oligohydraminos/terr	No h/o nicu admission	consanginious	100/70mmhg	48/min	altered	<3SEC	2.066	3.1	4	HIGH RIS	3	HIGH RIS	24 days	discharged sucessfully
6year	Female	372576	C/o fever since 5 days, c/o cough since 5 days, a	Not signif	Not Significant	BOOKED CASE	Term / lscs / cried at birth	No h/o nicu admission	consanginious	80/40mmhg	68/min	altered	>3SEC	99	0.5	5	HIGH RIS	3	HIGH RIS	10 days	Death
7 months	Female	397193	C/o fever since 3 days; c/o 1 episode of convulsio	Significar	Not Significant	BOOKED CASE	Term/LSCS/cried at birth	No h/o nicu admission	consanginious	100/60mmhg	40/min	altered	<3SEC	3.889	3.8	4	HIGH RIS	2	HIGH RIS	4 days	discharged sucessfully
6 years	Male	24887	C/o fever 1 week; c/o 2 episode of convulsions	Significar	Not Significant	BOOKED CASE	FTNVD/cried at birth	No h/o nicu admission	consanginious	100/80mmhg	20/min	intact	<3SEC	1.9	0.8	1	LOW RIS	1	LOW RIS	2 days	discharged sucessfully
7 years	Female	393182	C/o fever and cough since 5 days; c/o 4 episodes	Significar	Not Significant	NOT A BOOKED CA	FTNVD/cried at birth	No h/o nicu admisssion	non consanginious	100/70mmhg	22/min	intact	>3SEC	3.59	1.68	3	LOW RIS	0	LOW RIS	3 days	discharged sucessfully
5 years	Female	381995	C/o convulsions 5 episodes; c/o fever since today	Significar	Not Significant	BOOKED CASE	Late preterm/cried at birth/r	No h/o nicu admission	consanginious	110/60mmhg	72/min	altered	>3SEC	0.61	1.1	2	LOW RIS	1	LOW RIS	5 days	discharged sucessfully
10 years	Male	14682	C/o fever since 3 days; c/o 1 episode of convulsio	Not signif	Not Significant	BOOKED CASE	Term/aga/cried at birth	No h/o nicu admission	consanginious	110/70mmhg	20/min	intact	<3SEC	0.6	1.16	2	LOW RIS	2	HIGH RIS	2 days	discharged sucessfully
3 years	Male	31462	C/o fever since 2 days; c/o convulsions 2 episode	Significar	Not Significant	BOOKED CASE	Term/NVD/cried at birth	No h/o nicu admission	non consanginious	90/60mmhg	32/min	altered	<3SEC	1.2	0.8	3	LOW RIS	3	HIGH RIS	5 days	discharged sucessfully
1year 2 m	Male	54110	C/o fever; c/o convulsions 2 episodes	Significar	Not Significant	BOOKED CASE	FTNVD/cried at birth	No h/o nicu admission	non consanginious	100/60mmhg	38/min	altered	<3SEC	17.06	2	4	HIGH RIS	2	HIGH RIS	3 days	discharged sucessfully
6 YEARS	Female	218830	c/o fever since 2 days and convulsion since 2	Significa	Not Significant	BOOKED CASE	late preterm/AGA/DIDNOT	H/O NICU ADMISSION FOR	non consanginious	100/70MMHG	28/MIN	altered	<3SEC	0.5	1.1	2	LOW	2	HIGH RIS	2 DAYS	discharged sucessfully
MONTH	Female	61382	C/O 1 EPISODE OF CONVULSION C/O FEVER S	significa	Not Significant	BOOKED CASE	IMMEDIATELY AT	NO H/O NICU ADMISSION	consanginious	80/50MMHG	40CPM	intact	<3SEC	9.92	2.64	3	RISK	1	LOW RIS	2 DAYS	discharged sucessfully
5 YEARS	Male	61406	CONVULSION 2 EPISODES	significa	Not Significant	BOOKED CASE	IMMEDIATELY AT	NO H/O NICU ADMISSION	non consanginious	110/80	42/MIN	altered	<3SEC	30.2	2.82	4	RISK	2	HIGH RIS	6 DAYS	discharged sucessfully
MONTH	Male	88746	C/O FEVER SINCE 3 DAYS AND CONVULSION 3 EPISODE	Not significa	Not Significant	BOOKED CASE	LATE PRETERM/AGA/LSCS/CI B	H/O NICU ADMISSION/ H/O HYPERBILIRUBINEMIA	consanginious	70/40MMHG	60/MIN	altered	<3SEC	99.9	3.6	5	RISK	3	HIGH RIS	12 DAYS	Death
6 YEARS	Female	87270	C/O FEVER SINCE 3 DAYS C/O VOMITING 2 DAYS AND 1 EPISODE CONVULSION	Not significa	Not Significant	BOOKED CASE	TERM/AGA/LSCS/CIAB	NO H/O NICU ADMISSION	non consanginious	100/70MMHG	20/MIN	altered	<3SEC	3.2	2.6	5	HIGH RISK	2	HIGH RIS	6 DAYS	discharged