CORRELATION OF URINARY URIC ACID, CREATININE RATIO WITH THE SEVERITY OF HYPOXIC ISCHEMIC ENCEPHALOPATHY

By

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Dissertation submitted to

SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION AND RESEARCH, TAMAKA, KOLAR, KARNATAKA

in partial fulfillment of the requirement for the degree of

DOCTOR OF MEDICINE (M.D)

IN

PAEDIATRICS

Under the Guidance of

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APRIL 2019

SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION &RESEARCH, TAMAKA, KOLAR, KARNATAKA

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Department	PAEDIATRICS
Acceptable Maximum Limit	10%
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Submission Date	2018-12-07 04:11:54

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ACKNOWLEDGEMENT

One of the joys of completion of this dissertation is to look over the journey past and remember and thank all the people who have helped and supported me along this long but fulfilling road. First and foremost, I thank the Almighty for giving me the strength and ability to carry out this study.

I am deeply indebted and grateful to my guide, ***Dr. BEEREGOWDA. Y.C.*** *Professor of Department of Paediatrics, Sri Devaraj Urs Medical College, for his able guidance, support, timely advice and constant encouragement throughout the period of the study.*

I am deeply indebted and grateful to my co guide, ***Dr. SHASHIDHAR K.N.*** *Professor of Department of Biochemistry, Sri Devaraj Urs Medical College, for his able guidance, support, timely advice and constant encouragement throughout the period of the study.*

I thank ***Dr. K. N. V PRASD****, Professor and HOD of Department of Paediatrics, Sri Devaraj Urs Medical College, for his support and guidance during my post graduation.*

I thank ***Dr. SUDHAREDDY .V.R*** *Professor of Department of Paediatrics, Sri Devaraj Urs Medical College, for his support and guidance during my post graduation*

I thank ***Dr. KRISHNAPPAA .J*** *Professor of Department of Paediatrics, Sri Devaraj Urs Medical College, for his support and guidance during my post graduation*

I thank ***Dr. BHANUCHAND.P*** *Professor of Department of Paediatrics, Sri Devaraj Urs Medical College, for his support and guidance during my post graduation*

*I would also like to warmly extend my gratitude to **Dr. SHRUTHI APPAJI**, Senior Resident, Department of Paediatrics, Sri Devaraj Urs Medical College, for her constant encouragement.*

*No words can express the gratitude I feel towards my beloved parents, **KOTA SUBBARAO** and **VIJAYALAKSHMI**, whose countless sacrifices and endless love has made me who I am today in life.*

*I also thank my sister, **Chaitanya** for her motivation and for being a constant source of strength.*

*I am thankful to my postgraduate colleague, **Dr. Ritesh .V** my seniors and dear juniors for all their love, motivation and help.*

I am truly blessed in having the most wonderful friends and would like to thank them for their endless support.

I will be failing my duty if I do not thank all my patients involved in this study, without whose co-operation and patience this study would have been impossible.

Dr. K.V. SUMAN

LIST OF ABBREVIATIONS USED

ABG	Arterial Blood Gas Analysis
APGAR	Activity, Pulse, Grimace, Appearance, Respiration
ATP	Adenosine Triphosphate
BP	Blood Pressure
CO	Cardiac Output
CK-BB	Creatine Kinase Brain Bound
CT	Computed Tomography
CVP	Central Venous Pressure
DWI	Diffusion-Weighted Imaging
EEG	Electro Encephalogram
HIE	Hypoxi Ischemic Encephalopathy
IQR	InterQuartile Range
LSCS	Lower Segment Caesarean Section
MRI	Magnetic Resonance Imaging
NICU	Neonatal Intensive Care Unit
PPHN	Persistent Pulmonary Hypertension of the Newborn
PROM	Premature Rupture of Membranes
PMRS	Proton Magnetic Resonance Spectroscopy
NSE	Neuron Specific Enolase
XO	Xanthine Oxidase

ABSTRACT

"CORRELATION OF URINARY URIC ACID, CREATININE RATIO WITH THE SEVERITY OF HYPOXIC ISCHEMIC ENCEPHALOPATHY"

BACKGROUND:

Perinatal asphyxia is a common neonatal problem and contributes significantly to neonatal morbidity and mortality. Perinatal asphyxia leading to HIE may result in adverse effects on all major body systems. Many of these complications are potentially fatal. So, if the adverse effects of hypoxia on the newborn are considered, there is a need to identify infants who will be at high risk for hypoxic ischemic encephalopathy and progressing to different stages of HIE that is deteriorating need to prevent early neonatal death as a consequence of HIE.

A variety of markers have been examined to identify hypoxic injury .Though there are more studies for understanding mechanisms leading to birth asphyxia, studies for early determination of brain tissue damages due to birth asphyxia are still lacking. The urinary uric acid creatinine ratio as a predictor of mortality and severity of disease in HIE, and has established the correlation between the APGAR score and the urinary uric acid to creatinine ratio. Moreover, rising of urinary uric acid and creatinine ratio as a predictor of HIE in critically ill infants.

OBJECTIVES:

1. To estimate urinary uric acid and creatinine levels in spot urine sample
2. To correlate the urinary uric acid, creatinine ratio with severity of hypoxic ischemic encephalopathy.

MATERIALS AND METHODS:

One Hundred and Eight asphyxiated babies were enrolled this observational study in R. L. Jalappa Hospital and Research Centre, Department of Paediatrics attached to Sri Devaraj Urs Medical College Kolar during January 2017 to May 2018. They were further sub-grouped as HIE1, HIE2 and HIE3. Urinary Uric acid and creatinine were measured spectrophotometrically.

RESULTS:

Among the 108 asphyxiated babies, 36 (33.30%) participants had HIE 1, 40 (37%) had HIE 2 and 32 (29.60%) had HIE 3. The mean urine uric acid was 48.7 ± 40.69 in the asphyxiated babies. Range between was 1.60 to 216 (95% CI 40.94 to 56.46). The median urine creatinine

was 15 units in the study SUBJECTS, with an IQR (10 - 29.75). The median Urine Urea creatinine ratio was 2.20, with an IQR (1.51 - 3.18) .Among the HIE 1 babies, the median urine uric acid was 34 (17.25, 75.75). It was 38.20 (20.75, 57.50) people with HIE 2 and it was 35.50 (28, 60.45) among the people with HIE 3. The difference in the urine uric acid across HIE was statistically not significant (p 0.864). Among the HIE 1 babies, the median urine creatinine was 23.25 (14, 45.75). it was 16.90 (10, 31.25) people with HIE 2 and it was 10 (7.55, 15.05) among the people with HIE 3. The difference in the urine creatinine across HIE was statistically significant (p <0.001). Among the HIE 1 people, the median ratio was 1.26 (1.06, 2.20). It was 2.07 (1.87, 2.50) people with HIE 2 and it was 3.47 (2.85, 4.57) among the people with HIE 3. The difference in the ratio across HIE was statistically significant (p <0.001).

CONCLUSION:

Early identification of infants at highest risk for developing hypoxic ischemia is essential for a timely intervention and management of asphyxiated babies. In the absence of perinatal records which was found in many of my cases our study, it is difficult to retrospectively diagnose perinatal asphyxia. Infants with asphyxia have higher urinary uric acid to creatinine ratio. It might be used as an indicator for assessment of severity of birth asphyxia. So urinary uric acid to creatinine ratio can be used as an additional non-invasive, specific, early, easy and cost effective biochemical marker of perinatal asphyxia which biochemically supports the clinical diagnosis of sarnat and sarnat staging. The results of this study indicate that the urinary ratio of uric acid to creatinine may be used as an additional marker of perinatal asphyxia in term babies and premature infants where need to be studied yet. In comparison with other markers it is a simple, quick, and inexpensive way to detect hypoxic stages in a neonatal NICU after birth. So we conclude that urinary uric acid and creatinine ratio concentration increase considerably at different stages of HIE. Hence, urinary uric acid and creatinine ratio can be used as prognostic marker in different stages of HIE.

KEY WORDS: Birth asphyxia, hypoxic ischemic encephalopathy, urinary uric acid creatinine ratio

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Introduction

INTRODUCTION

Perinatal asphyxia is a common neonatal problem and contributes significantly to neonatal morbidity and mortality. Globally, hypoxia of the newborn (birth asphyxia) or the foetus ("fresh stillbirth") is estimated to account for 23% of the 4 million neonatal deaths and 26% of the 3.2 million stillbirths each year [1]. An estimated 1 million children who survive birth asphyxia live with chronic neurodevelopmental morbidities, including cerebral palsy, mental retardation, and learning disabilities. [1] Perinatal asphyxia leading to HIE may result in adverse effects on all major body systems. Many of these complications are potentially fatal. In a term infant with perinatal asphyxia renal, neurological, and cardiac and lung dysfunction occurs in 50%, 28%, 25% and 23% cases respectively Neonatal hypoxia is one of the leading causes of neonatal mortality in developing countries.[1] So, if the adverse effects of hypoxia on the newborn are considered, there is a need to identify infants who will be at high risk for hypoxic ischemic encephalopathy and early neonatal death as a consequence of perinatal hypoxia.[2]

A variety of markers have been examined to identify perinatal hypoxia including low APGAR scores, cord pH, electroencephalograms (EEG), computed tomography (CT) and magnetic resonance imaging (MRI) scans and Doppler flow studies .Though there are more studies for understanding mechanisms leading to birth asphyxia, studies for early determination of tissue damages due to birth asphyxia are still lacking. Increasing the activity of xanthine oxidase (XO) during the inflammatory process and neonatal sepsis has already been studied. Hypoxic ischemic events and inflammatory processes can cause renal damage, which leads to urinary uric acid excretion, which is a product of purine catabolism and free radicals due to the activation of xanthine oxidase .[3]Previous studies [1-2]have evaluated the Urinaryuric acid to creatinine ratio as a predictor of mortality and severity of disease in perinatal asphyxia, and has established the correlation between the APGAR score and the urinary uric acid to creatinine ratio.Moreover, rising urine uric acid and creatinine ratio as a predictor of mortality in critically ill infants.[4]

PERINATAL ASPHYXIA:

Perinatal asphyxia is a condition which occurs in during second and third stages of labor in which impaired gases exchange takes place leading to hypoxemia, acidosis and hypercarbia.Fetal hypoxemia can be measured by cord blood ph .[5]

Neonatal encephalopathy is a clinical and not an etiologic term that describes an abnormal neurobehavioral state consisting of an altered level of consciousness (including hyper alert state) and usually other signs of brainstem and/or motor dysfunction.

Hypoxic-ischemic (HI) brain injury refers to neuropathology attributable to hypoxia and/or ischemia as evidenced by neuroimaging, magnetic resonance imaging, computed tomography [CT]) or pathologic abnormalities. Biochemical markers of brain injury such as creatinekinase brain bound (CK-BB) and neuron specific enolase (NSE) are not used routinely in clinical practice .[6]The diagnosis of HIE and/or HI brain but ruling out other etiologies of neurologic dysfunction is a critical part of the diagnostic evalution.

There is a lack of certainty regarding the timing or severity of asphyxia in many cases.

Etiologies of hypoxia-ischemia may be multiple and include the following:

1. Maternal factors: hypertension (acute or chronic), hypotension, infection (including chorioamnionitis), hypoxia from pulmonary or cardiac disorders, diabetes, maternal vascular disease, and *in utero* exposure to cocaine.
2. Placental factors: abnormal placentation, abruption, infarction, fibrosis, or hydrops
3. Uterine rupture
4. Umbilical cord accidents: prolapse, entanglement, true knot, compression
5. Abnormalities of umbilical vessels

6. Fetal factors: anemia (e.g., from fetal-maternal hemorrhage), infection, cardiomyopathy, hydrops, severe cardiac/circulatory insufficiency
7. Neonatal factors: cyanotic congenital heart disease, persistent pulmonary hypertension of the newborn (PPHN), cardiomyopathy, other forms of neonatal cardiogenic and/or septic shock, respiratory failure due to meconium aspiration syndrome, neonatal pneumonia, pneumothorax, or other etiologies

PATHOPHYSIOLOGY

These include the following:

1. Decreased O2 delivery to the fetus from reduced placental blood flow.
2. Increased O2 consumption in both mother and fetus.

Hypoxia-ischemia causes a number of physiologic and biochemical alterations:

1. Mild elevation in blood pressure (BP), an increase in central venous pressure (CVP), and essentially no change in cardiac output (CO). This is accompanied by a redistribution of CO with an increased proportion going to the brain, heart, and adrenal glands (diving reflex). When there is severe but brief asphyxia (e.g., placental abruption then stat cesarian section), it is thought that this diversion of blood flow to vital deep nuclear not occur, hence results in the typical pattern of injury to the sub cortical and brainstem nuclei.
2. With **prolonged asphyxia**, there CO_2 vasoreactivity. This, in turn, may lead to further disturbances in cerebral perfusion, particularly when there is cardiovascular involvement with hypotension and/or decreased CO, results in anaerobic metabolism and eventual cellular energy failure due to increased glucose utilization glycogen, phosphocreatine, and adenosine triphosphate (ATP).

Cellular dysfunction occurs as a result of diminished oxidative phosphorylation and ATP production. This energy failure impairs ion pump function, causing

accumulation of intracellular Na^+, Cl^-, H_2O, and Ca^{2+}; extracellular K^+; and excitatory neurotransmitters (e.g, glutamate). Impaired oxidative phosphorylation can occur during the primary HI insult(s) as well as during a secondary energy failure that usually begins approximately 6 to 24 hours after the initiating insult.

1. **Delayed neuronal death**
2. **Reperfusion of hypoxic tissue**

DIAGNOSIS

A. **Perinatal assessment of risk** includes awareness of preexisting maternal or fetal problems that may predispose to perinatal asphyxia and of changing
B. **Low APGAR scores** and need for resuscitation in the delivery room are common but nonspecific findings.

Many features of the APGAR score relate to cardiovascular integrity and **not** neurologic dysfunction resulting from asphyxia.[7]

1. APGAR score ≤3 for ≥10 minutes includes depression from maternal anesthesia or analgesia, trauma, infection, cardiac or pulmonary disorders, neuromuscular, and other central nervous system disorders or malformations.[8]
2. If the APGAR score is >6 by 5 minutes, perinatal asphyxia is not likely.[9]

The diagnosis of neonatal encephalopathy includes a number of etiologies in addition to perinatal hypoxia-ischemia. Asphyxia may be suspected and HIE reasonably included in the differential diagnosis when there is:

1. Prolonged (>1 hour) antenatal acidosis
2. Fetal HR <60 beats per minute
3. APGAR score ≤3 at ≥10 minutes
4. Need for positive pressure ventilation for >1 minute or first cry delayed >5 minutes

5. Seizures within 12 to 24 hours of birth
6. Burst suppression or suppressed background pattern on EEG or amplitude-integrated electroencephalogram (aEEG)

NEUROLOGIC SIGNS:

Encephalopathy: Newborns with HIE must have abnormal consciousness by definition, whether mild, moderate, or severe.

Brainstem and cranial nerve abnormalities:

Newborns with HIE may have brainstem dysfunction, which may manifest as abnormal or absent brainstem reflexes, including pupillary, corneal, oculocephalic, cough, and gag reflexes. There can be abnormal eye movements such as dysconjugate gaze, gaze preference, ocular bobbing or other abnormal patterns of bilateral eye fixation or blink to light. Newborns may show facial weakness (usually symmetric) and have a weak or absent suck and swallow with poor feeding. They can have apnea or abnormal respiratory patterns.

Motor abnormalities: With greater severity of encephalopathy, there is generally greater hypotonia, weakness, and abnormal .With severe HIE, primitive or grasp reflex may be diminished. Over days hyperreflexia if there is significant HI brain injury. Note that if a newborn shows significant hypertonia within the first day or so after birth, the HI insult may have occurred earlier in the antepartum period and have already resulted in established HI brain injury.

Seizures occur in up to 50% of newborns with HIE and usually start within 24 hours after the HI insult.

Seizures indicate that the severity of encephalopathy is moderate or severe, not mild.

1. Seizures may be subtle, tonic, or clonic. It can sometimes be difficult to differentiate seizures from jitteriness or clonus, although the latter two are usually suppressible with firm hold of the affected limb(s).
2. Because seizures are often subclinical (electrographic only) and abnormal movements or posture may not be seizure, EEG remains the gold standard for diagnosing neonatal seizures, particularly in HIE.
3. Seizures may compromise ventilation and oxygenation, especially in newborns who are not receiving mechanical ventilation. It is important to adequately support respiration to avoid additional hypoxic injury

MULTIORGAN DYSFUNCTION:

Other organ systems in addition to the brain usually exhibit evidence of asphyxial damage. In a minority of cases (<15%), the brain may be the only organ exhibiting dysfunction following asphyxia. In most cases, multiorgan dysfunction occurs as a result of systemic hypoxia ischemia.

A. The proximal tubule of the kidney is especially affected by decreased perfusion, leading to acute tubular necrosis with oliguria and a rise in serum creatinine.
B. **Cardiac** dysfunction is caused by transient myocardial ischemia. The electrocardiogram (may show ST depression in the midprecordium and T-wave inversion in the left precordium. Echocardiographic findings include decreased left ventricular contractility, especially of posterior wall; elevated ventricular end-diastolic pressures; tricuspid insufficiency; and pulmonary hypertension. In severely asphyxiated newborns, dysfunction more commonly affects the right ventricle. A fixed HR may indicate severe brainstem injury.
C. **Pulmonary** effects include increased pulmonary vascular resistance leading to PPHN.

D. **Gastrointestinal** effects include an increased risk of bowel ischemia and necrotizing enterocolitis

LABORATORY EVALUATION OF ASPHYXIA

A. **Cardiac evaluation**

B. Neurologic markers of brain injury

1. Serum CK-BB may be increased in asphyxiated newborns within 12 hours of the insult but has not been correlated with long-term neurodevelopmental outcome. CK-BB is also expressed in placenta, lungs, GI tract, and kidneys. Other serum markers such as protein S-100, NSE, and urine markers have been measured in newborns with asphyxia and HIE.
2. In practice, serum and urine markers of brain injury are not routinely used to evaluate for the presence of brain injury or to predict outcome.

C. Renal evaluation

1. Blood urea nitrogen and serum Cr may be elevated in perinatal asphyxia. Typically, elevation is noted 2 to 4 days after the insult.
2. Fractional excretion of Na^+ or renal failure index may help confirm renal insult.
3. Urine levels of β2-microglobulin have been used as an indicator of proximal tubular dysfunction, although not routinely. This low molecular weight protein is freely filtered through the glomerulus and reabsorbed almost completely in the proximal tubule.

BRAIN IMAGING

A. **Cranial sonographic** examination

B. **CT** may be used to detect cerebral edema, hemorrhage, and eventually HI brain injury.

C. **MRI.** These conventional sequences are best for the detection of brain injury at least 7 to 10 days, and a scan as late as 14 days or older may be needed to show the full extent of the injury, particularly if early MRI shows less injury than suspected by clinical exam or EEG findings.

1. **Diffusion-weighted imaging (DWI)**

   Early DWI scans will usually show restricted diffusion in brain regions affected by hypoxia-ischemia. At 7 to 10 days of age, there is pseudonormalization of diffusion, so DWI can appear normal despite the presence of HI injury. After 7 to 10 days, diffusion is usually increased in regions of HI brain injury. Hypothermia appears to delay the time to

   pseudonormalization of diffusion.

2. **Proton magnetic resonance spectroscopy (MRS)**, also called *proton-MRS* or *H-MRS,*[10] measures the relative concentrations of various metabolites in tissue. Elevated lactate,decreased N-acetylaspartate,and alterations of the ratios of these two metabolites in relation to choline or creatine can indicate HIE and help with determining neurologic prognosis.
3. **Magnetic resonance (MR) angiography or venography** may occasionally be useful if there is suspicion of vascular anomalies, thromboembolic disease, or sinus venous thrombosis, which can occasionally be found in association with HIE.

EEG is used to both detect and monitor seizure activity and also to define abnormal background patterns such as discontinuous, burst suppression, low voltage, or isoelectric patterns.. This method consists of a reduced montage with one- or two-channel EEG with parietal electrodes. Although aEEG may detect some seizures, there are data showing that aEEG detects far fewer seizures compared with

conventional EEG and that the quality of aEEG interpretation depends very much on the experience and expertise of the reader.

PATHOLOGIC FINDINGS OF BRAIN INJURY:

A. Specific neuropathology may be seen after moderate or severe HIE.

1. Most common type of injury seen following perinatal asphyxia is selective neuronal necrosis.

In the term newborn, more severe, prolonged HI insults result in bilateral parasagittal cortical and subcortical white matter injury.

2. Focal or multifocal cortical necrosis result in cystic encephalomalacia and/or ulegyria

B. Neuropathology may reflect the type of asphyxial episode, although the precise pattern is not predictable.

1. Prolonged partial episodes of asphyxia tend to cause diffuse cerebral (especially cortical) necrosis, although there is often involvement of subcortical ± brainstem structures as well.
2. Acute total asphyxia, when relatively brief, affects primarily the brainstem, thalamus, and basal ganglia and tends to spare the cortex in large part, except for the perirolandic cortex.
3. Partial prolonged asphyxia followed by a terminal acute asphyxial event (combination) is probably present in most cases

Aims and objectives

AIMS AND OBJECTIVES

1. To estimate urinary uric acid and creatinine levels in spot urine sample
2. To correlate the urinary uric acid, creatinine ratio with severity of hypoxic ischemic encephalopathy

Review of literature

REVIEW OF LITERATURE

Chen HJ, Yau KI, Tsai KS in 2000 conducted a study the results of this study indicate that the ratio of urinary uric acid to cratinine may be used as an additional marker of perinatal asphyxia in term and premature infants. In comparison with other markers such as xanthine, hypoxanthine, and ascorbic acid, it is a simple, quick, and inexpensive way to detect hypoxic episodes in a neonatal intensive care unit within 24 hours after birth. In a study shown that the urinary uric acid/creatinine ratio was found to be a good, early, simple and reliable screening test for the early diagnosis and assessment of perinatal asphyxia.

Pallab Basu, Sabyasachi Som, Nabendu Choudhuri and Harendranath Das in 2008, conducted a randomized case control study was conducted over 12 months time on 31 asphyxiated and 31 normal newborn to see whether urinary uric acid can be used as a marker of perinatal asphyxia and can be correlated with the clinical diagnosis by APGAR score. Uric acid and creatinine were estimated in spot urine within 24 hours after birth in both cases and controls. A ratio between concentrations of uric acid to creatinine was estimated and compared between cases and controls. It was found urinary uric acid to creatinine ratio can be used as an additional non-invasive , easy and at the same time early biochemical marker of birth asphyxia which biochemically supports the clinical diagnosis and severity grading of asphyxia by APGAR score[12]

Deepak Kumar, Partha Kumar Chaudhari, Anil Kumar Chaudhary, Shipra Kamal in 2016 conducted a study on 110 neonates comprising 55 cases and 55 controls born in Rajendra Institute of Medical Sciences. Spot urine sample collected within first day of life. A cut-off urinary uric acid to creatinine urinary uric acid to

creatinine ratio ratio value of >1.14 was taken as the cut-off level. They have found that The urinary uric acid to creatinine ratios were found to be higher in asphyxiated infants (2.58±1.09) when compared with those in the controls (0.86±0.17; p<0.001). The cut-off urinary uric acid to creatinine ratio value of >1.14 has 84% sensitivity with a specificity of 94% and has a positive predictive value of 93.33%with negative predictive value of 85.45%with an accuracy of 89%.they have concluded that the urinary uric acid/creatinine ratio was found to be a good, early, simple and reliable screening test for the early diagnosis and assessment of perinatal asphyxia.[1]

Sally Palit *et al,* 2013 conducted a observational, cross-sectional study to assess for a possible correlation between blood PH and urinary uric acid creatinine ratio in asphyxiated newborn in Prof. Dr. R. D. Kandou Hospital, Manado, North Sulawesi, from November 2013 to April 2014. Subjects were term newborns with asphyxia. Blood pH and urinary uric acid to creatinine ratio were compared with Pearson's correlation test. Forty subjects were included in the study. Their predominant risk factor for asphyxia was fetal distress. Subjects' mean blood pH was 7.1 (SD 0.1) and mean urinary uric acid urine creatinine ratio was 3.7 (SD 1.9). There was a moderate negative correlation between blood pH and urinary UUA/UCr ratio (r= -0.55; P<0.001) and they have concluded that newborns with asphyxia, lower blood pH is correlated with higher urinary uric acid urine creatinine ratio.[13]

Aparna Varma Bhongir, Akhil Varma Venkata Yakama, Subhajit Saha, Sejal B. Radia, Jayalakshmi Pabbati1 in 2015 conducted a study, it revealed the mean urinary uric acid and creatinine ratio (2.58± 0.48 vs 1.89 ± 0.59) is significantly higher in asphyxiated group than in the control group. The umbilical cord blood pH had significant positive correlation with 1st minute APGAR score (r= 0.41, p=0.003) 5th minute APGAR (r= 0.44, p=0.002), while urinary uric acid to creatinine ratio ratio

had significant negative correlation with cord blood pH (r= -0.63, p=0.002). Urinary uric acid to creatinine ratio with criterion of >2.43 had 80% sensitivity, 87.5% specificity with area under curve (AUC) of 0.84 (p=0.003) had a better predictive value. Urinary uric acid to creatinine ratio is easy, non-invasive, painless and economical adjuvant parameter with better predictive value for diagnosing perinatal asphyxia with simple diagnostic equipment[14]

Shahin Nariman *et al* in 2014 conducted a study total of 362 preterm infants with a mean gestational age of 32.7 weeks were admitted to the NICU, out of whom 64 (17.6%) had severe disease and 43 (11.8%) died. The mean Urinary uric acid to creatinine ratio was significantly higher in the admitted neonates (P = 0.0001). There was a negative correlation between the Urinary uric acid to creatinine ratio and the 1-minute APGAR score (r = -0.17, P= 0.006) and the 5-minute APGAR score (r = -0.19, P = 0.003). The 1-minute APGAR scores were negatively correlated with the outcome (OR = 0.68; P < 0.001) and the duration of stay (= -.28; P < 0.001). There was no significant correlation between 5-minute APGAR scores and the outcome. There was a significant positive correlation between the Urinary uric acid to creatinine ratio and an unfavourable outcome (P = 0.006) and increasing duration of stay (P = 0.009). [2]

Kattupalli. Yashwanth *et al* in 2016 conducted a study in Niloufer Hospital attached to Osmania Medical College. Total 100 babies were included in the study. Abnormal neurological examination is significantly more in cases when compared to Controls with P< 0.001. Among the 100 neonates in case group, 40 (40%) had no seizures. 60 (60%) had seizures as an abnormal neurological examination finding.Abnormal neurological examination is significantly more (60%) in cases when compared to Controls with P<0.001. Among the 100 neonates in the case group, 40 (40%) had

mild HIE, 42(42%) had moderate HIE and 18 (18%) had severe HIE during the course in NICU. Cases and control were randomly selected, 100 cases (Neonatal Asphyxia) and control of 100 cases (normal newborns), were undertaken in assessing the Urinary Acid / Creatinine ratio as a marker in neonatal Asphyxia, the correlation of of urinary uric acid and creatinine ratio with HIE status among the cases and it was found to be statistically significant with a p value of < 0.001. They have also concluded that the cut-off Urinary uric acid and creatinine ratio value of >1.4 has 94% sensitivity with a specificity of 96% and has a positive predictive value of 95.52% with a negative predictive value of 94.12% with an accuracy of 95%.

Elango Krishnana, Venmugil Ponnusamy, Sathiya Priya Sekar in 2017 conducted a prospective case control study in KAVP Govt Medical College. 100 asphyxiated and non asphyxiated babies were included and they have found that mean Urinary uric acid and creatinine ratio in the cases and controls groups were 2.58±1.09 and 0.86±0.17 respectively. The ratio also correlated well with the stage of HIE. They have concluded that the ratio of Urinary uric acid to creatinine ratio enables early and rapid recognition of asphyxial injury and also the assessment of its severity and the potential for short term morbidity or death.[16]

Kinjal Prahaladbhai Patel, Mayur Goradhanbhai Makadia, Vishwal Indravardan Patel, Haridas Neelakandan Nilayangode, in 2017 conducted a case control study at teaching hospital in central Gujarat to assess the urinary uric acid/creatinine ratio as an additional marker for perinatal asphyxia compared with ABG analysis in APGAR score monitoring. They concluded that the clinical diagnosis of asphyxia by APGAR scores be supported by other investigations so that early decision can be taken about the level of care the baby needs. pH, lactates and base deficits change with establishment of respiration following resuscitation.

However, pH, lactate, base deficit estimations are invasive and need rapid estimations. non-invasive urinary uric acid to creatinine ratio may be an answer to these issues as it easy, economical and equally efficient.[17]

Srikrishna *et al* in 2017 conducted a randomized case control study in Rajarajeshwri Medical College and Hospital and they have taken 50 asphyxiated and 50 normal newborn. urinary uric acid and creatinine were estimated in spot urine within 24 hours after birth in both cases and controls. Ratio between the concentrations of uric acid to creatinine was estimated and comparison done between cases and controls. They have found that urinary uric acid to creatinine ratio can be used as an additional non-invasive, easy and early biochemical marker of birth asphyxia which biochemically supports the clinical diagnosis and the severity grading of asphyxia by APGAR score. They have concluded that urinary uric acid to creatinine ratio can be used as an additional non-invasive, easy and early biochemical marker of birth asphyxia which biochemically supports the clinical diagnosis and the severity grading of asphyxia by APGAR score.[18]

Ioannis B *et al* conducted a meta-analysis in 2018 in which fourteen studies were included in the study and investigated 1,226 neonates. Urinary uric acid to creatinine ratio was significantly higher in neonates with perinatal asphyxia than in healthy controls. They have concluded that Urinary uric acid to creatinine ratio is a rapid and easily detected biomarker that may help physicians identify hypoxic-ischemic encephalopathy. However, large-scale prospective studies are still needed to determine its value in predicting mortality, as well as short and long-term adverse neurological outcomes.[19]

Methodology

MATERIALS

The study design was an observational study. The study was conducted in joint collaboration with Department of Paediatrics and Department of Biochemistry in Sri Devaraj Urs Medical College and Hospital, Kolar, Karnataka. The study protocol was approved by the Institutional ethical committee.

INCLUSION CRITERIA:

Babies with hypoxic ischemic encephalopathy as per SARNATH and SARNATH staging criteria.

EXCLUSION CRITERIA

Suspected babies with inborn errors of metabolism, babies with major congenital malformations were excluded from study.

METHODS OF DATA COLLECTION:

Detailed maternal history, birth events, APGAR score, sex of the baby and anthropometry of the baby were recorded on the proforma. Gestational age is assessed by New Ballard scoring system. Thorough clinical and neurological examination was done for all the neonates included in the study. The babies who has suffered asphyxia and as per standard staging system (SARNAT AND SARNAT) which fit in to the criteria of HYPOXIC ISCHEMIC ENCEPHALOPATHY will be categorized in to stage 1, stage 2 and stage 3 were included in to the study group. The asphyxiated neonates were monitored for all components of SARNAT AND SARNAT staging in the immediate neonatal period in the NICU. Grading system used to grade the severity of HIE was SARNAT and SARNAT staging 1976.[20]

Stage	Stage1(MILD)	Stage2(Moderate)	Stage3(Severe)
Level of consciousness	Hype alert, irritable	Lethargic or obtunded	Stuporous, comatose
Neuromuscular control:	Uninhibited, over reactive	Diminished spontaneous movement	Diminished or absent spontaneous movement
Muscle tone	Normal	Mild hypotonia	Flaccid
Posture	Mild distal flexion	Strong distal flexion	Intermittent decerebration
Stretch reflexes	Overactive	Overactive, disinhibited	Decreased or absent
Segmental myoclonus	Present or absent	Present	Absent
Suck	Uninhibited, over reactive	Diminished spontaneous movement	Diminished or absent spontaneous movement
Moro	Normal	Mild hypotonia	Flaccid
Oculovestibular	Normal	Overactive	Weak or absent
Tonic neck	Slight	Strong	Absent
Autonomic function:	Generalized sympathetic	Generalized parasympathetic	Both systems Depressed
Pupils	Mydriasis	Miosis	Midposition, often unequal; poor light reflex
Respirations	Spontaneous	Spontaneous; occasional apnea	Periodic; apnea
Heart rate	Tachycardia	Bradycardia	Variable
Seizures	None	Common focal or multifocal (6-24 hours of age)	Uncommon (excluding decerebration)
Electroencephalographic Findings	Normal (awake)	Early: generalized low voltage, slowing (continuous delta and theta) Later: periodic pattern (awake); seizures focal or multifocal; 1.0-1.5 Hz spike and wave	Early: periodic pattern with isopotential phases Later: totally Isopotential
Duration of symptoms	<24 hours	2-14 days	Hours to weeks
Outcome	About 100% normal	80% normal; abnormal if symptoms more than 5-7 days	About 50% die; remainder with severe sequelae

SAMPLING PROCEDURE:

All deliveries conducted in our hospital are attended by residents trained in neonatal resuscitation few of them are outside born babies referred to our NICU for further management whose APGAR status not known. One hundred and eight neonates were recruited for the study. 108 term babies with >37 weeks of gestations both inborn and out born babies were admitted to NICU with APGAR score of 6 or less at 5 minutes of birth and with history of baby did not cried immediately after birth were included, and monitored strictly and babies which were progressed to HIE I , HIE II ,HIE III are taken in to the study. The neonates that were included in the study did not undergo therapeutic hypothermia before the collection of the urinary sample. The urinary samples were collected as early as possible baby diagnosed clinically to have HIE and all urine samples were collected by staff nurse in sterilized disposable urine bag and analyzed in hospital laboratory for Urinary uric acid to creatinine ratio. Urinary uric acid was estimated by auto analyzer by spectrophotometric uricase method. Urinary creatinine was estimated in same above instrument by using sarcosine oxidase method. Ethical clearance was obtained by Institutional Ethics Committee.

STATISTICAL ANALYSIS:

Qualitative data is presented in the form of proportions and pie diagrams, bar charts used. Quantitative data is presented as mean, standard deviation. Descriptive analysis was carried out by mean and standard deviation for quantitative variables, frequency and proportion for categorical variables. Data was also represented using appropriate diagrams like bar diagram, pie diagram and box plots.

All Quantitative variables were checked for normal distribution within each category of explanatory variable by using visual inspection of histograms and normality Q-Q

plots. Shapiro-Wilk test was also conducted to assess normal distribution. Shapiro-Wilk test p <0.05 was considered as normal distribution.

For none normally distributed Quantitative parameters, Medians and Interquartile range (IQR) were compared across the study groups using Kruskal Wallis test (> 2 groups).

P < 0.05 was considered statistically significant. IBM SPSS version 22 was used for statistical analysis.[20] Data was analyzed using IBM SPSS 22 VERSION software.

STATISTICAL METHODS:

Urine uric acid, urine creatinine and Urine Urea creatinine ratio were considered as primary outcome variable. Study group HIE (1,2,3) was considered as primary explanatory variable.

SAMPLE SIZE CALCULATION:

Sample size was estimated based on the outcome of Urinary Uric Acid Creatinine Ratio in the hypoxic encephalopathy subjects from the study by Deepak Kumar *et al*[1], considering the mean ± SD of 2.58±1.09, at 5% alpha error and at 95% Confidence level sample size of 45 was obtained.

Formula used:

$$\text{Sample size} = \frac{Z_{1-\alpha/2}{}^2 SD^2}{d^2}$$

$Z_{1-\alpha/2}$ = Is standard normal variate as mentioned in previous section.
SD = Standard deviation of variable. Value of standard deviation can be taken from previously done study or through pilot study.
d = Absolute error or precision as mentioned in previous section

Z = at 5% alpha error = 1.96

SD = 1.09

d= 5% error

Results

RESULTS

A total one hundred and eight people were included in the analysis.

Table 1: Descriptive analysis of age in hours during the presentation to the NICU in study subjects.

Parameter	Mean ± SD	Median	Min	Max	95% C.I	
					Lower	Upper
Age presentation hrs	13.67 ± 19.24	6.00	0.27	144.00	10.00	17.34

The mean age at presentation was 13.67 ± 19.24 hours in the study subjects, ranged between 0.27 to 144 hours (95% CI 10 to 17.34).

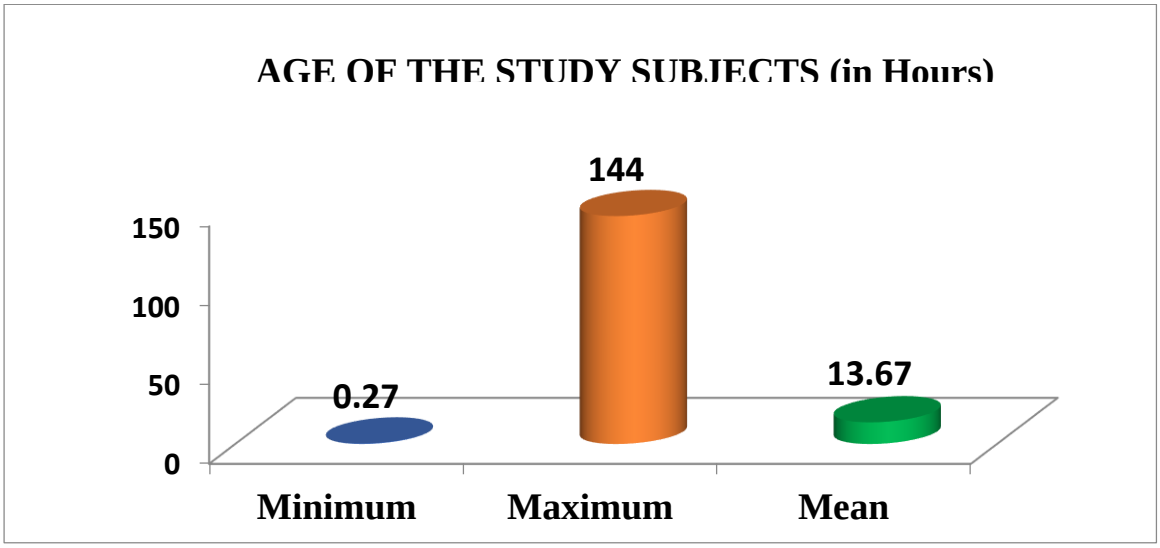


Figure 1: Bar diagram showing age in hours distribution of subjects

Table 2: Gender distribution of subjects between two groups

Gender	Frequency	Percentages
Male	62	58%
Female	46	43%

Among the studysubjects, 62 (57.40%) subjects were male and 46 (42.60%) were female.

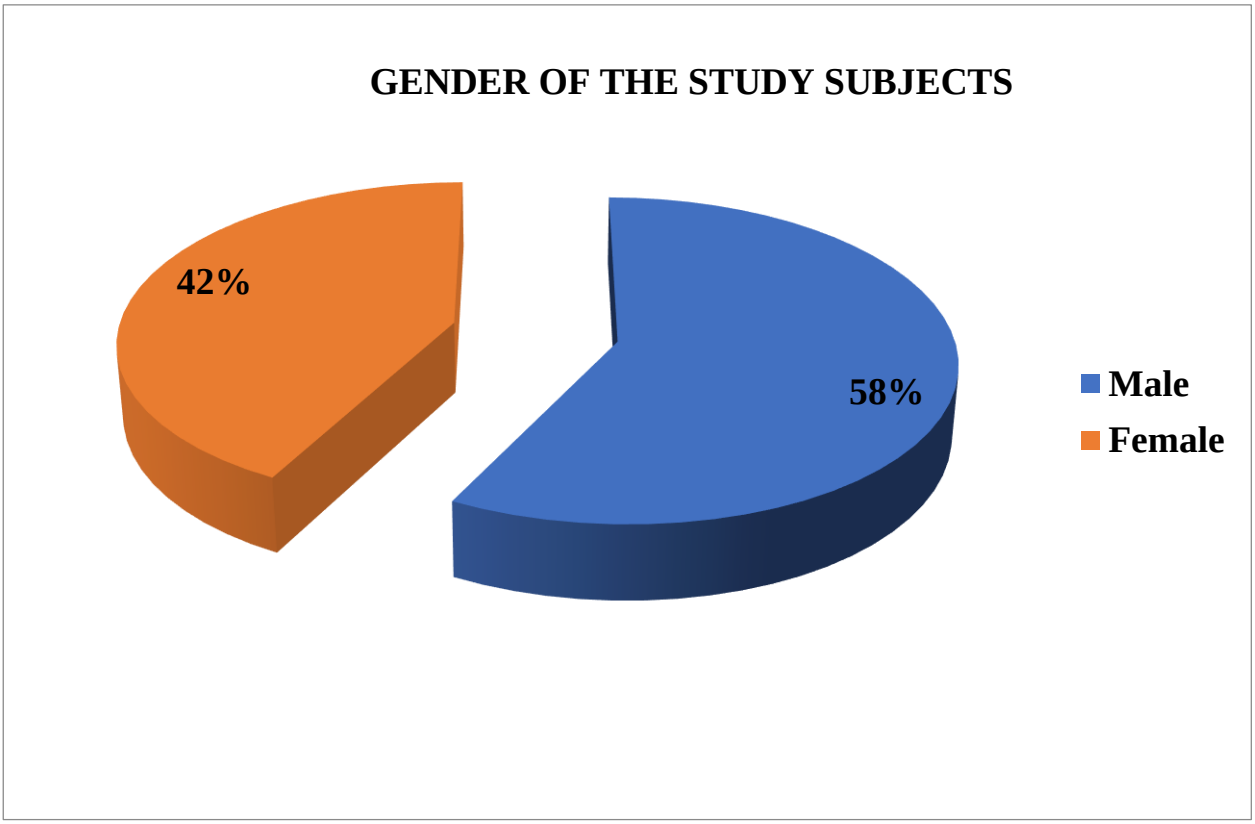


Figure 2: Pie diagram showing gender distribution of subjects

Table 3: Descriptive analysis of consanguinity in study subjects (N=one hundred and eight)

Consanguinity	Frequency	Percentages
Not Present	93	85%
2nd degree	11	11%
3rd degree	4	4%

Among the study subjects, 11 (11%) subjects had 2nd degree consanguinity and 4 (4%) had 3rd degree consanguinity and no consanguinity in 93 (85%).

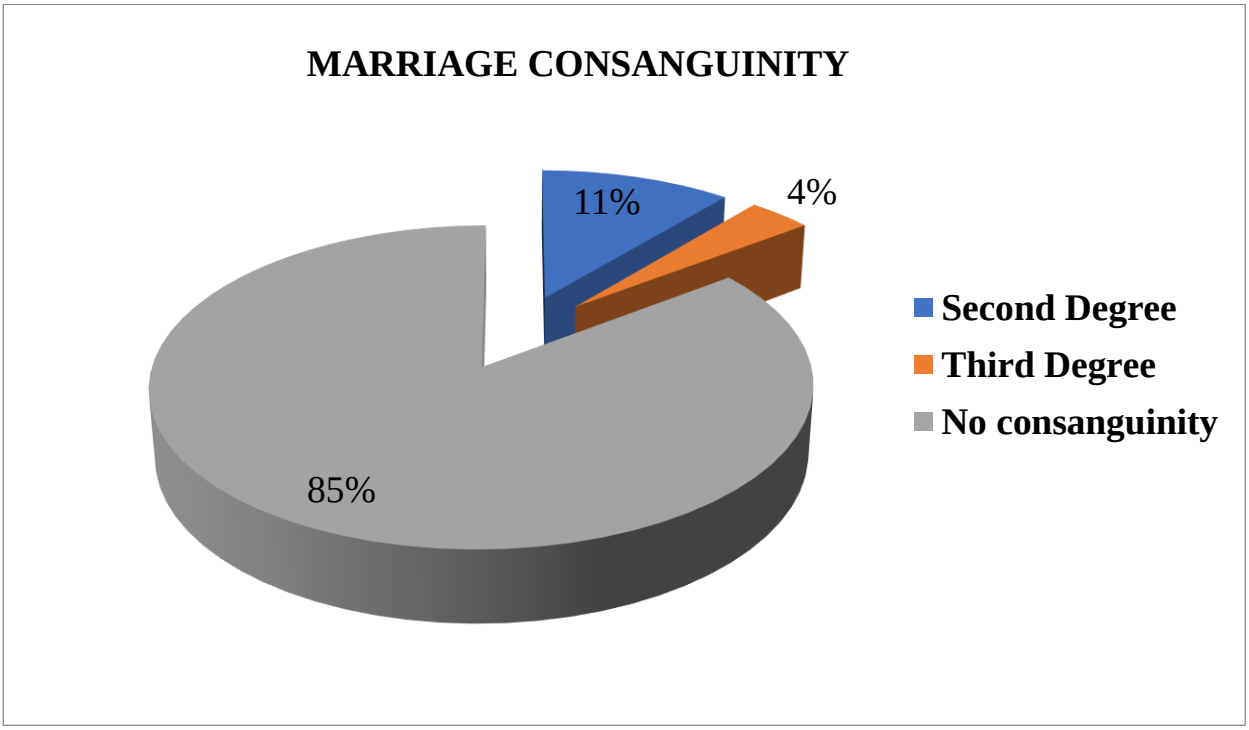


Figure 3: Pie diagram showing age in hours distribution of subjects

Table 4: Descriptive analysis of Gestational age in days in study subjects (N=one hundred and eight)

Number	Minimum	Maximum	Mean±SD
One hundred and eight	37	41.4	39.22±1.06

The mean gestational age was 39.22 ± 1.06 in the study subjects. Range between was 37 weeks to 41.4 weeks (95% CI 39.1 to 39.60).

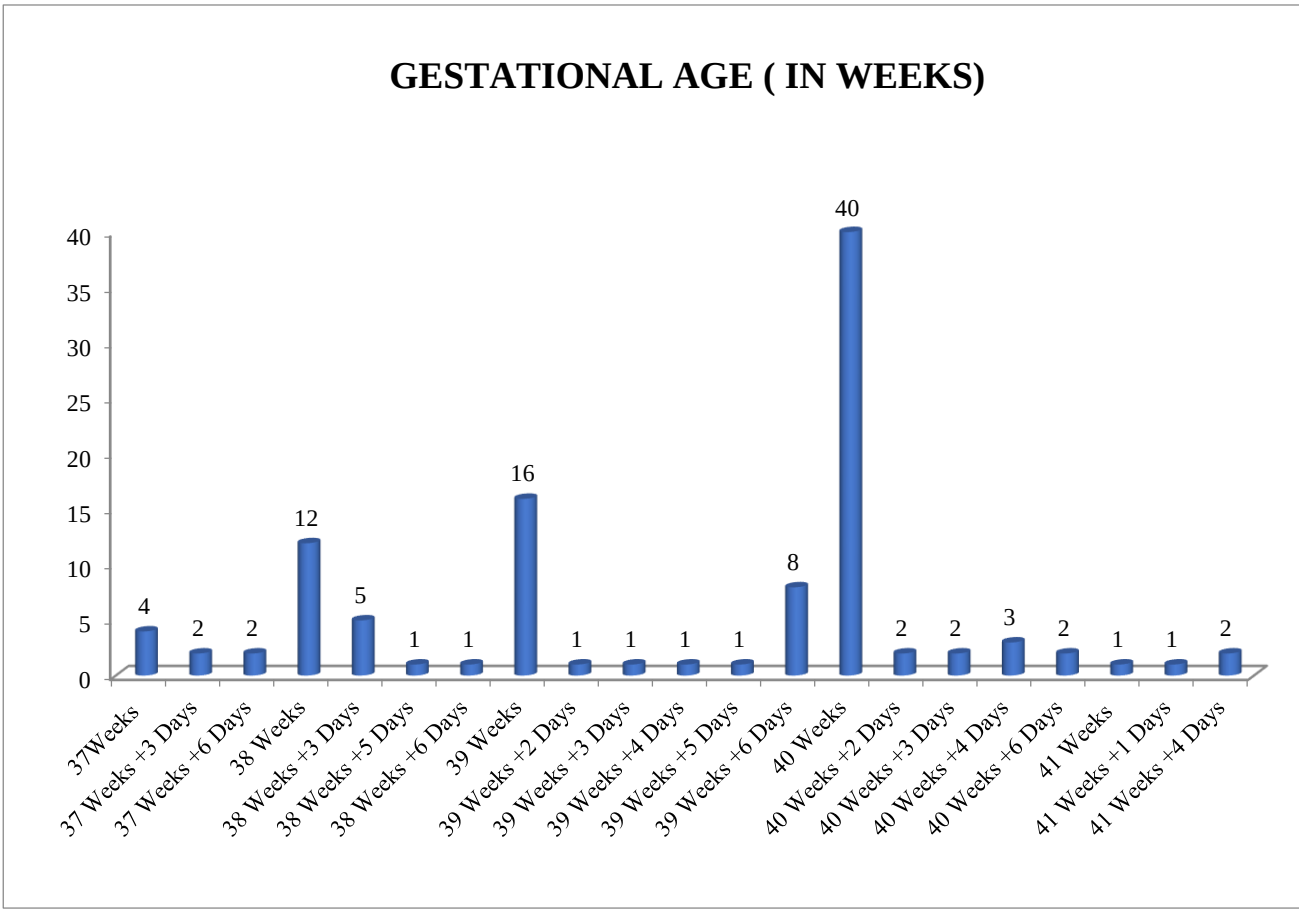


Figure 4: Bar diagram showing gestational age in weeks distribution of subjects

Table 5: Descriptive analysis of mode of delivery in the study subjects (N=one hundred and eight)

Mode of delivery	Frequency	Percentage
Normal vaginal delivery	68	63.00%
LSCS	33	30.60%
Forceps assisted delivery	4	3.70%
Vacuum assisted vaginal delivery	3	2.80%

Among the study subjects, the number of women with Mode of delivery was normal vaginal delivery in 68 (63%), LSCS in 33 (30.60%), forceps assisted delivery in 4 (3.70) and vacuum assisted in 3 (2.80%).

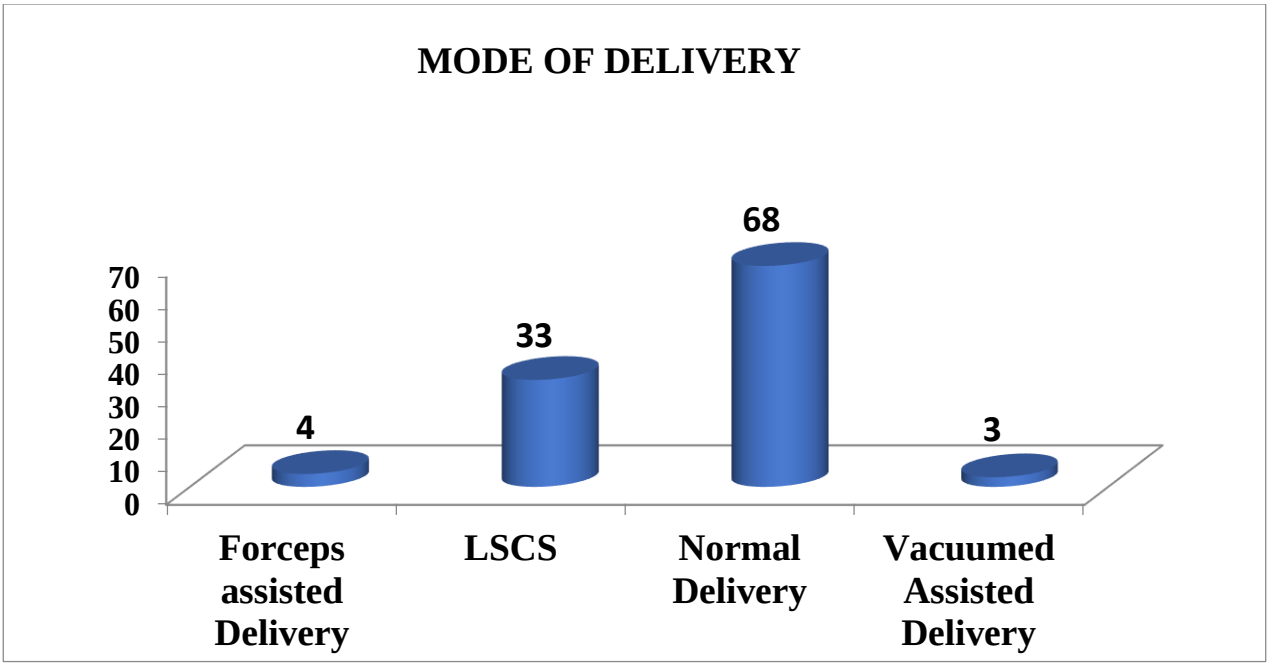


Figure 5: Bar diagram showing age in hours distribution of subjects

Table 6: Descriptive analysis of term/post-dated in the study subjects (N=one hundred and eight)

Term/post term/ post dated	Frequency	Percentages
Post dated	1	0.90%
Term	107	99.10%

Among the study subjects, only 1 (0.90%) participants had posted dated and 107 (99.10) had term. Post term nil

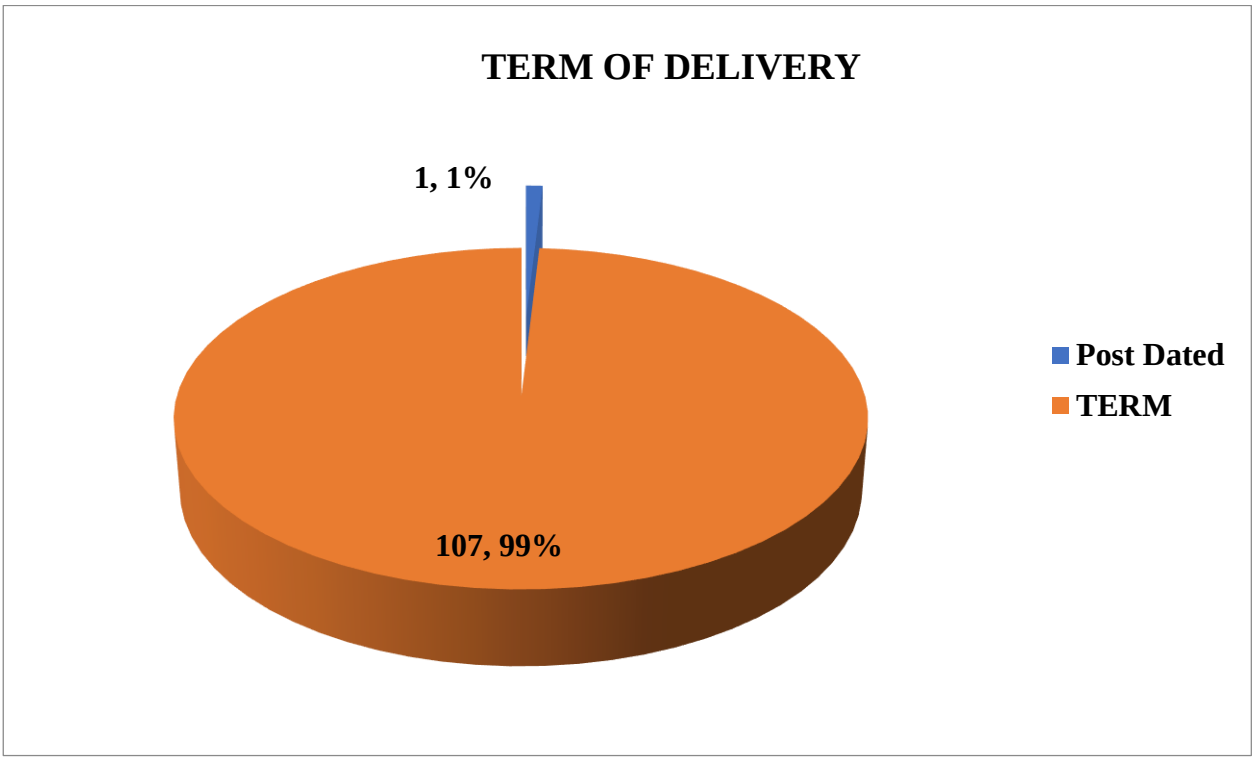


Figure 6: Pie diagram showing distribution of gestation in study subjects

Table 7: Descriptive analysis of pre-eclampsia in the study subjects (N=one hundred and eight)

Parameters	Frequency	Percentages
Pre-Eclampsia		
Present	5	4.60%
Nil	103	95.40%
Eclampsia		
Present	3	2.80%
Nil	105	97.20%
Bleeding Diathesis		
Present	0	0%
Nil	one hundred and eight	100.00%
Antepartum haemorrhage		
Present	0	0%
Nil	one hundred and eight	100.00%
Meconium stained liquor		
Present	18	16.70%
Nil	90	83.30%
PROM		
Prom	3	2.80%
Nil	105	97.20%
Chorionamniotis		
Present	0	0%
Nil	one hundred and eight	100.00%

Among the study subjects, 5 (4.60%) participants had present pre-eclampsia. Among the study subjects, 3 (2.80%) participants had present eclampsia. Among the study subjects, 18 (16.70%) participants had present meconium stained liquor. Among the study subjects, 3 (2.80) participants had prom.

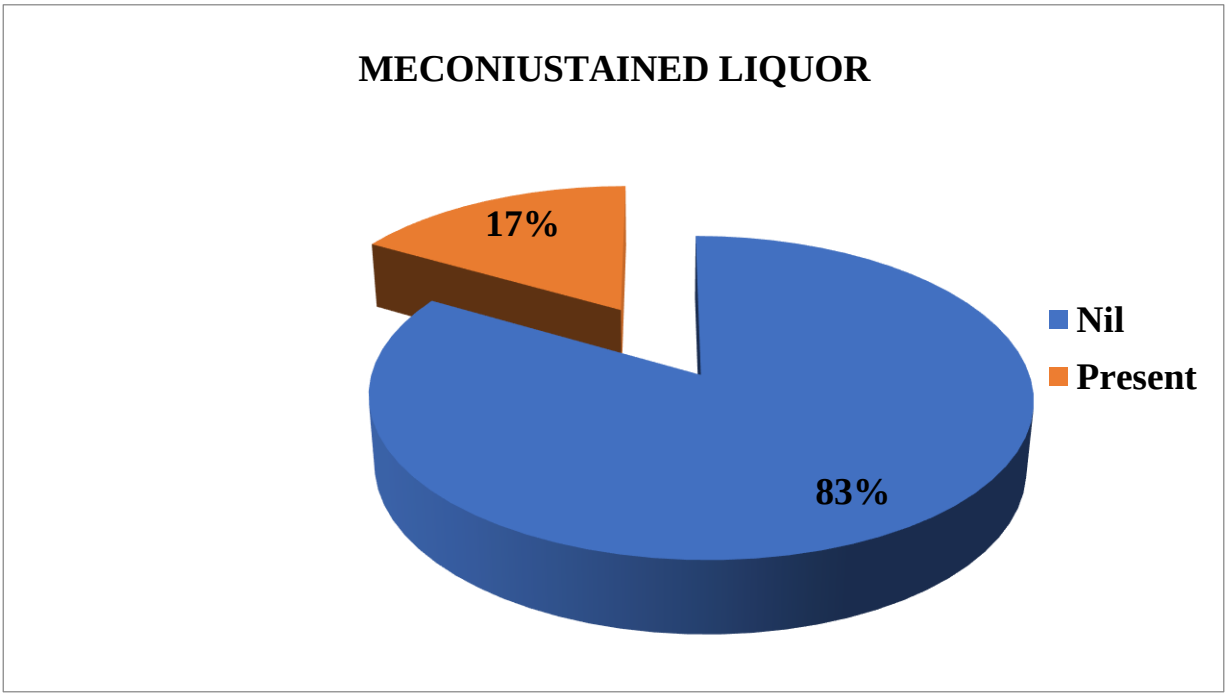


Figure 7: Pie diagram showing distribution of meconium stained liquor in study subjects

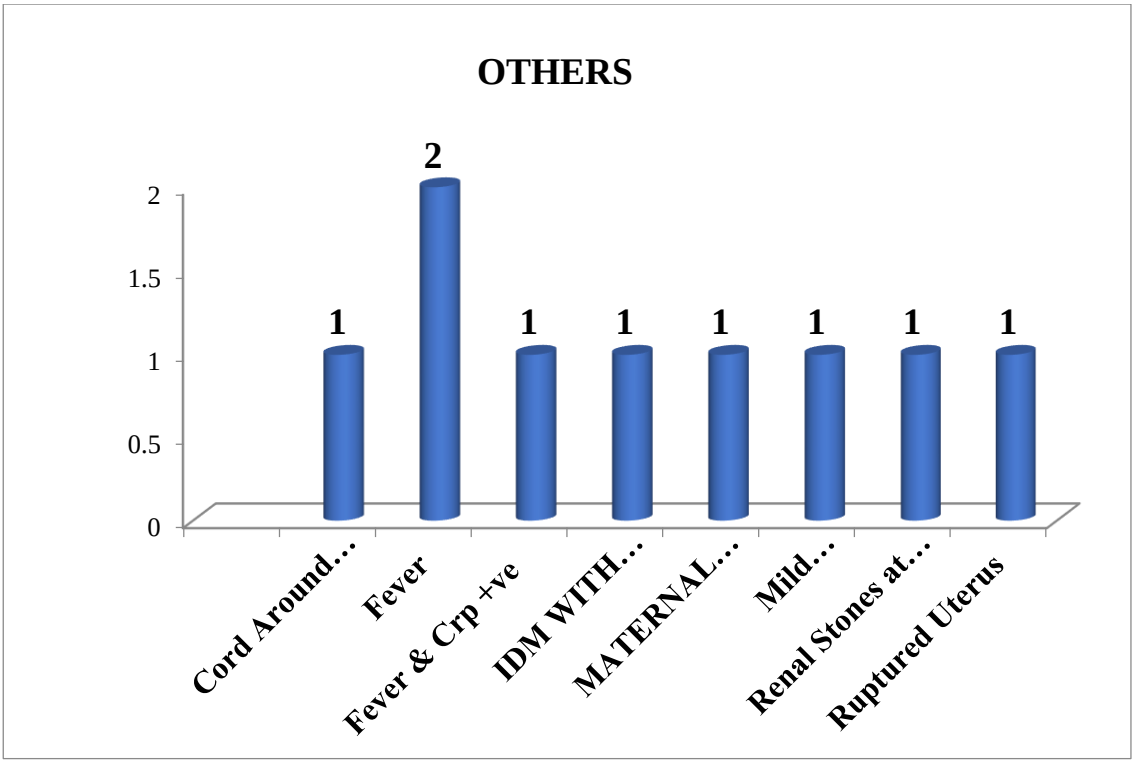


Figure 8: Bar diagram showing distribution of other maternal risk factors in which babies had developed HIE.

Table 8: Descriptive analysis of APGAR score in study subjects (N=59)

Parameter	Mean ± SD	Median	Min	Max	95% C.I	
					Lower	Upper
APGAR score 1 minutes	4.37 ± 1.11	4.00	1.00	7.00	4.08	4.66
APGAR score 5 minutes	6.25 ± 0.98	6.00	3.00	9.00	6.00	6.51
APGAR 10 minutes	7.09 ± 1.1	7.00	4.00	9.00	6.79	7.39

Note: Many children were born outside the current study setting and hence their data on APGAR score was not available. Hence we have analysed APGAR score for only the 59 children

The mean APGAR score at 1 minute was 4.37 ± 1.11 in the study subjects, minimum level was 1 and maximum level was 7 (95% CI 4.08 to 4.66). The mean APGAR score 5 minutes was 6.25 ± 0.98 in the study subjects, minimum level was 3 and maximum level was 9 (95% CI 6 to 6.51). The mean APGAR score 10 minutes was 7.09 ± 1.1 in the study subjects, minimum level was 4 and maximum level was 9 (95% CI 6.79 to 7.39).

Table 9 : Descriptive analysis of anthropometry in study subjects (N=one hundred and eight)

Parameter	Mean ± SD	Median	Min	Max	95% C.I	
					Lower	Upper
Birth weight in kg	2.88 ± 0.43	3.00	1.34	3.86	2.80	2.96
Head circumference in cm	33.11 ± 0.96	33.00	28.30	35.00	32.93	33.29
Length in cm	48.41 ± 1.91	48.00	38.00	52.00	48.05	48.78

The mean birth weight was 2.88 ± 0.43 in the study subjects, minimum weight was 1.34 kg and maximum weight was 3.86 kg (95% CI 2.80 to 2.96). The mean head circumference was 33.11 ± 0.96 in the study subjects, minimum level was 28.30 and maximum level was 35 (95% CI 32.93 to 33.29). The mean length was 48.41 ± 1.91 in the study subjects, minimum level was 38 and maximum level was 52 (95% CI 48.05 to 48.78).

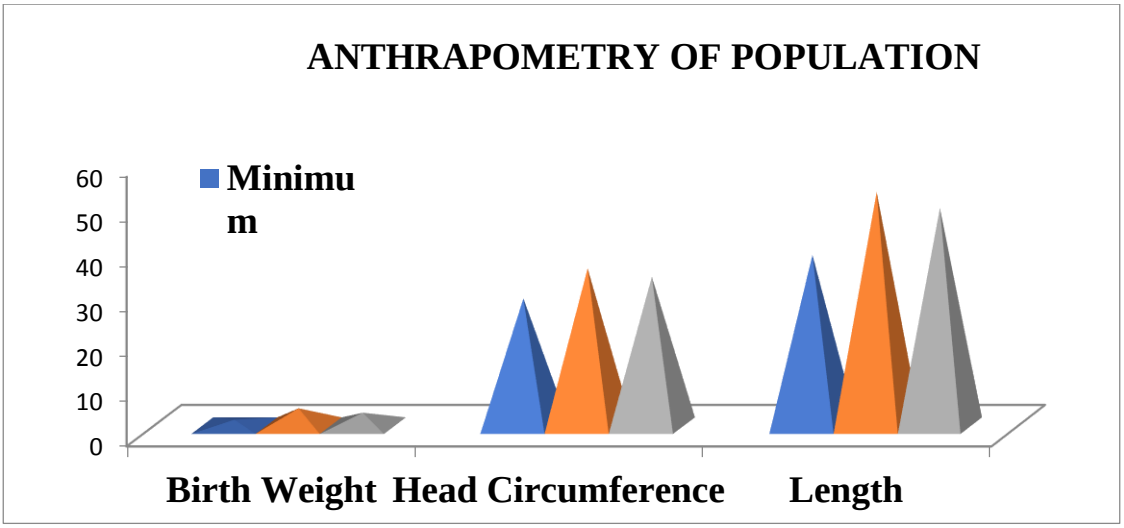


Figure 9: Bar diagram showing distribution of anthropometry in subjects.

Table 10: Descriptive analysis of general physical examination in the study subjects (N=one hundred and eight)

Parameter	Frequency	Percentages
Colour		
Pink	98	90.70%
Pale	4	3.70%
Cyanosis	6	5.60%
CRY		
Good	59	54.60%
Moderate	9	8.30%
Poor	40	37.00%
SUCK		
Good	57	52.80%
Moderate	9	8.30%
Poor	42	38.90%
Tone		
Good	57	52.80%
Moderate	9	8.30%
Poor	42	38.90%
Activity		
Good	58	53.70%
Moderate	9	8.30%
Poor	41	38.00%

Among the study subjects, 98 (90.70%) had pink colour, 4 (3.70%) had pale colour and 6 (5.60%) had cyanosis colour. Among the study subjects, 59 (54.60%) had good

cry, 9 (8.30%) had moderate and 40 (37%) had poor. Among the study subjects, 57 (52.80%) had good suck, 9 (8.30%) had moderate and 42 (38.90%) had poor. Among the study subjects, 57 (52.80%) had good tone, 9 (8.30%) had moderate and 42 (38.90%) had poor. Among the study subjects, 58 (53.70%) had good activity, 9 (8.30%) had moderate and 41 (38.00%) had poor.

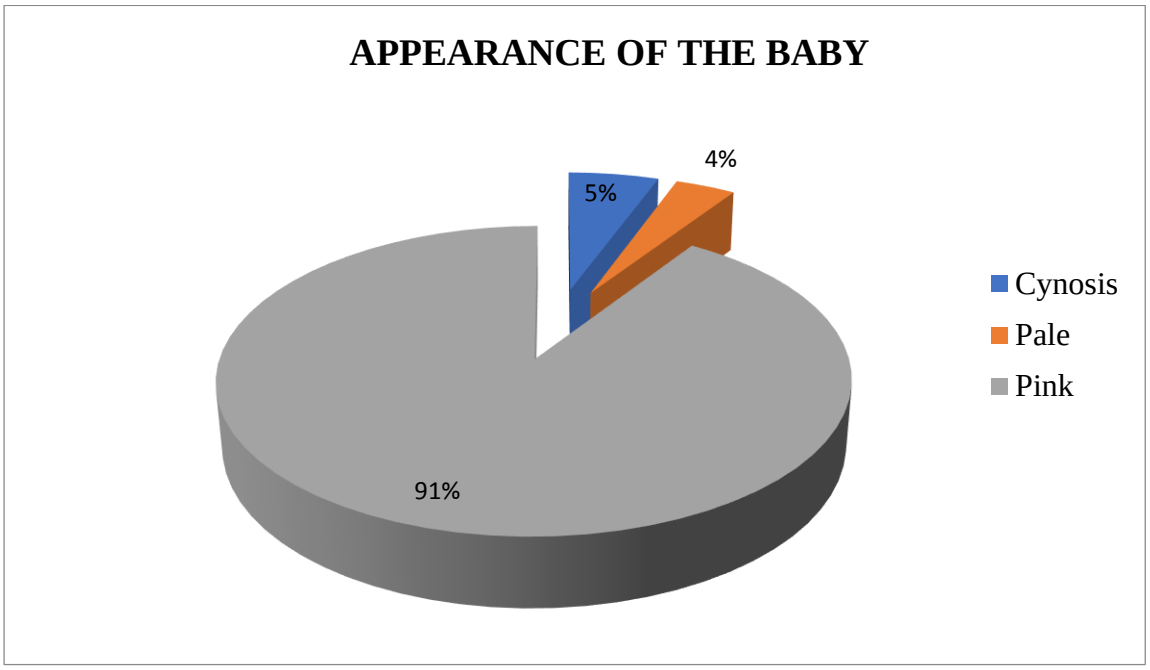


Figure 10: Pie diagram showing distribution of different type of appearances in study subjects

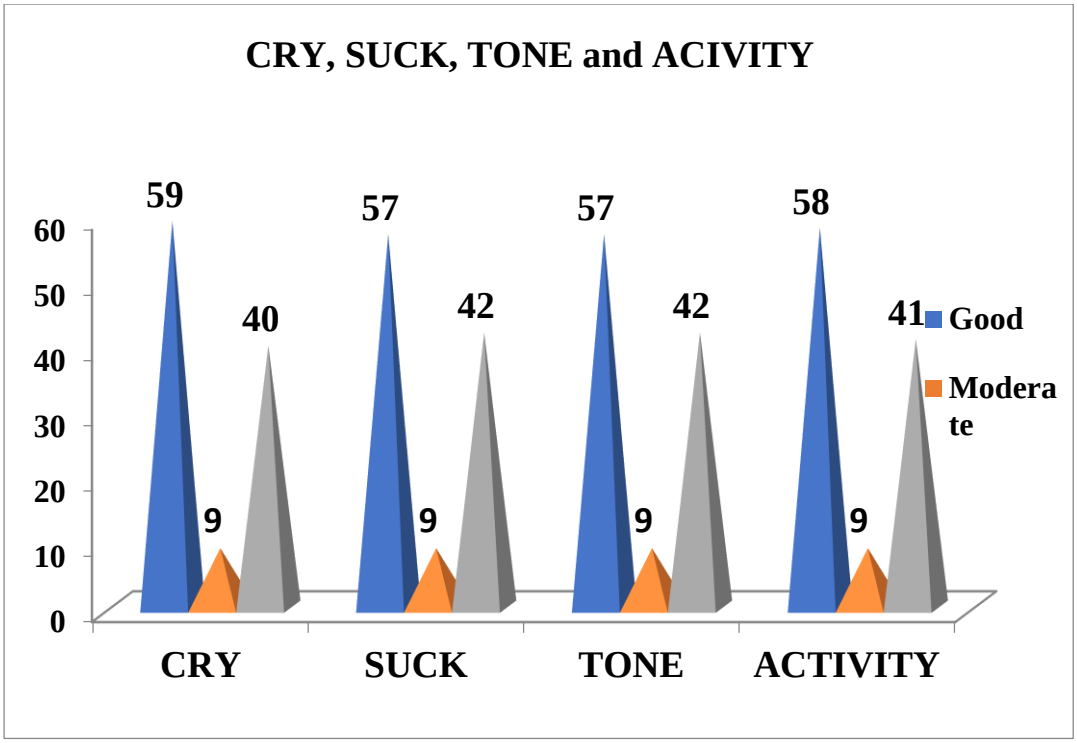


Figure 11: Bar diagram showing distribution of cry, suck, tone and activity in study subjects

Table 11: Descriptive analysis of heart rate in study subjects (N=one hundred and eight)

Parameter	Mean ± SD	Median	Min	Max	95% C.I	
					Lower	Upper
Heart rate	149.58 ± 24.05	154.00	10.00	220.00	145.00	154.17

The mean heart rate was 149.58 ± 24.05 in the study subjects. Minimum level was 10 and maximum level was 220 (95% CI 145 to 154.17).

Table 12: Descriptive analysis of cyanosis in the study subjects (N=one hundred and eight)

Cyanosis	Frequency	Percentages
Present	6	5.60%
Nil	102	94.40%

Among the study subjects, 6 (5.60%) subjects had present cyanosis.

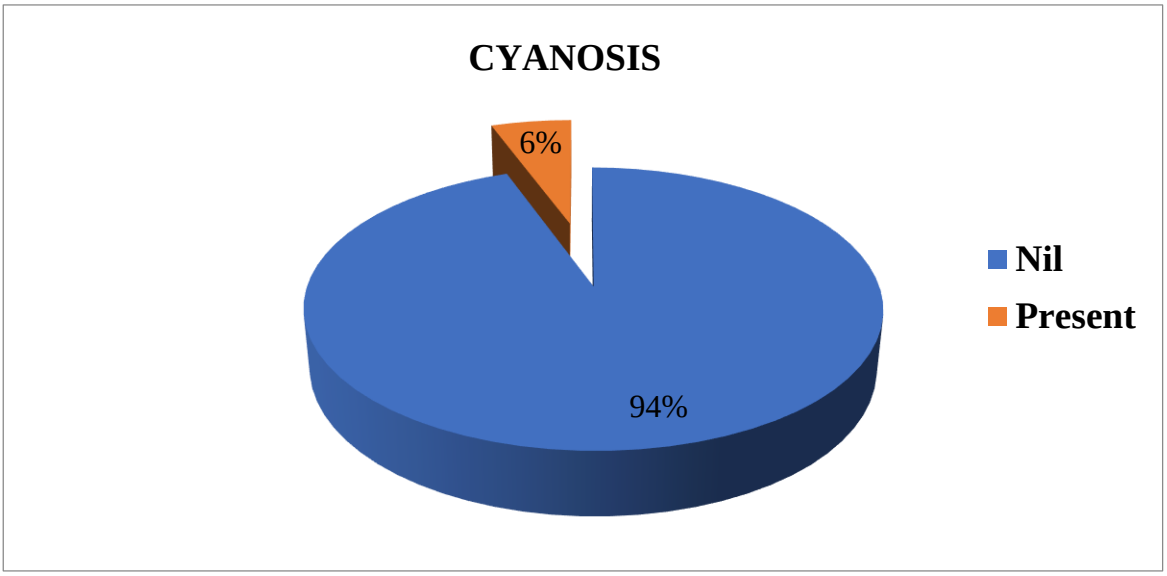


Figure 12: Pie diagram showing distribution of cyanosis in study subjects

Table 13: Descriptive analysis of HIE in the study subjects (N=one hundred and eight)

HIE	Frequency	Percentages
1	36	33.30%
2	40	37.00%
3	32	30%

Among the study subjects, 36 (33.30%) had HIE 1, 40 (37%) had HIE 2 and 32 (29.60%) had HIE 3.

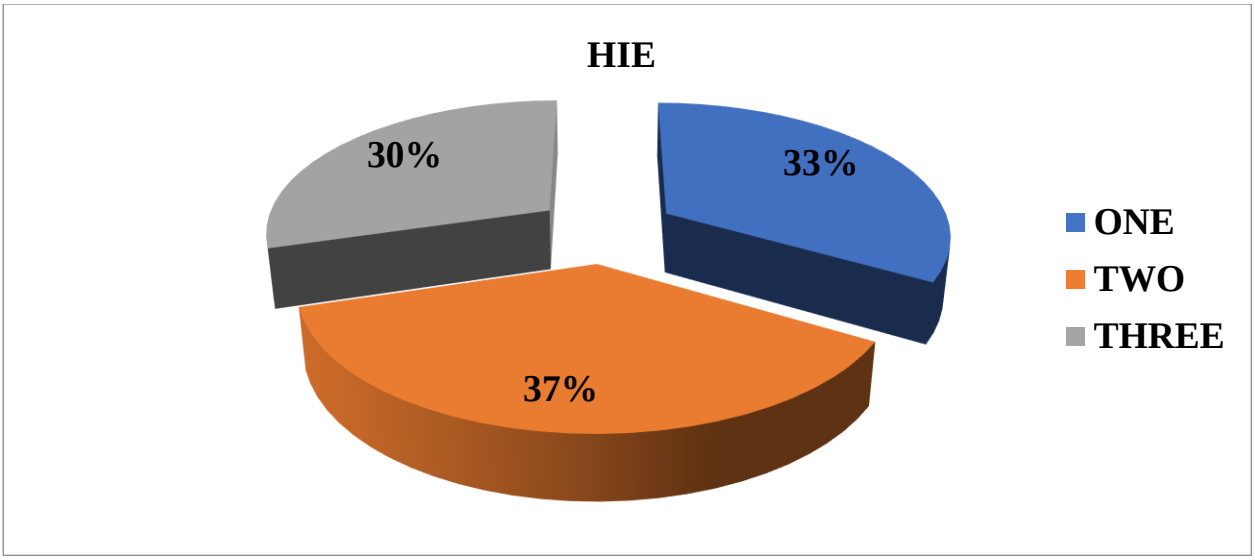


Figure 13: Pie diagram showing distribution of different HIE stages in study subjects

Table 14: Descriptive analysis of urine uric acid in study subjects (N=one hundred and eight)

Parameter	Mean ± SD	Median	Min	Max	95% C.I	
					Lower	Upper
Urine uric acid	48.7 ± 40.69	37.20	1.60	216.00	40.94	56.46

The mean urine uric acid was 48.7 ± 40.69 in the study subjects. Range between was 1.60 to 216 (95% CI 40.94 to 56.46).

Table 15: Descriptive analysis of urine uric acid in study subjects (N=one hundred and eight)

Parameter	Median	Inter quartile range (IQR)
Urine uric acid	37.20	(22.25, 61)

Table 16: Descriptive analysis of urine creatinine in study subjects (N=one hundred and eight)

Parameter	Mean ± SD	Median	Min	Max	95% C.I	
					Lower	Upper
Urine creatinine	25.14 ± 28.69	15.00	1.40	220.00	19.67	30.62

Table 17: Descriptive analysis of urine creatinine in study subjects (N=one hundred and eight)

Parameter	Median	Inter quartile range (IQR)
Urine creatinine	15	(10, 29.75)
Urine Uric acid/ creatinine ratio (UCR)	2.20	(1.51, 3.18)

The median urine creatinine was 15 units in the study subjects, with an interquartile range of 10 to 29.75. The median **Urine Uric acid creatinine ratio (UCR) was** 2.20, with an interquartile range of 1.51 to 3.18.

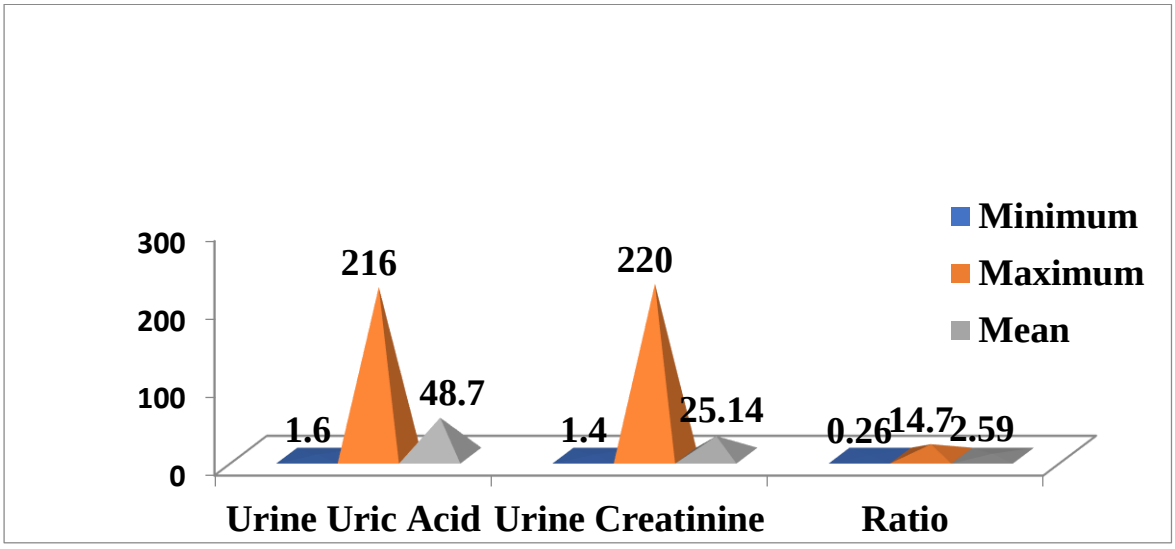


Figure 14: Bar diagram showing distribution of minimum, maximum and mean with respect to urinary uric acid, urinary creatinine and its ratio in study subjects.

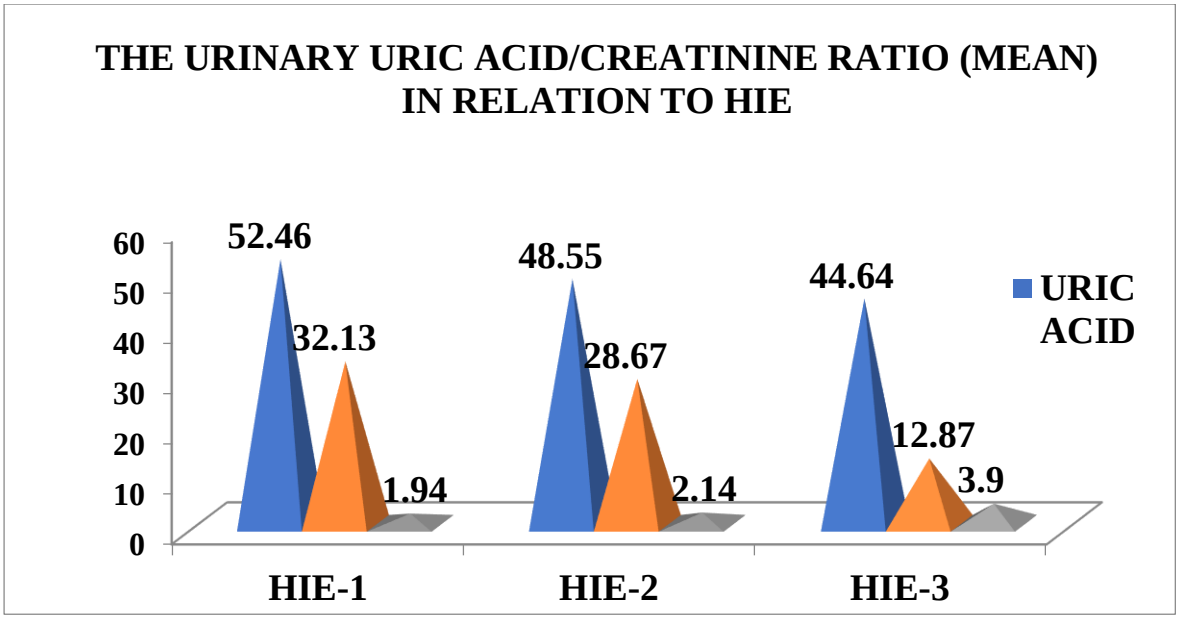


Figure 15: Bar diagram showing distribution of the urinary uric acid/creatinine ratio (MEAN) in relation to HIE in study subjects.

Table 18: Comparison of median URINE across the HIE (N=one hundred and eight)

Parameter	HIE			Kruskal Wallis P value
	1	2	3	
Urine uric acid Median (IQR)	34 (17.25, 75.75)	38.20 (20.75, 57.50)	35.50 (28, 60.45)	0.864
urine creatinine Median (IQR)	23.25 (14, 45.75)	16.90 (10, 31.25)	10 (7.55, 15.05)	<0.001
Ratio Median (IQR)	1.26 (1.06, 2.20)	2.07 (1.87, 2.50)	3.47 (2.85, 4.57)	<0.001

Among the HIE 1 subjects, the median urine uric acid was 34 (17.25, 75.75), it was 38.20 (20.75, 57.50) subjects with HIE 2 and it was 35.50 (28, 60.45) among the subjects with HIE 3. The difference in the urine uric acid across HIE was statistically not significant (P Value 0.864).

Among the HIE 1 subjects, the median urine creatinine was 23.25 (14, 45.75). it was 16.90 (10, 31.25) subjects with HIE 2 and it was 10 (7.55, 15.05) among the subjects with HIE 3. The difference in the urine creatinine across HIE was statistically significant (P Value <0.001). Among the HIE 1 subjects, the median ratio was 1.26 (1.06, 2.20). it was 2.07 (1.87, 2.50) subjects with HIE 2 and it was 3.47 (2.85, 4.57) among the subjects with HIE 3. The difference in the ratio across HIE was statistically significant (P Value <0.001). (Table 18 & Figure 17,18 and 19)

Figure 16: COMPARATIVE BOX PLOTS OF MEDIAN URINE URIC ACID ACROSS THE HIE (N=one hundred and eight)

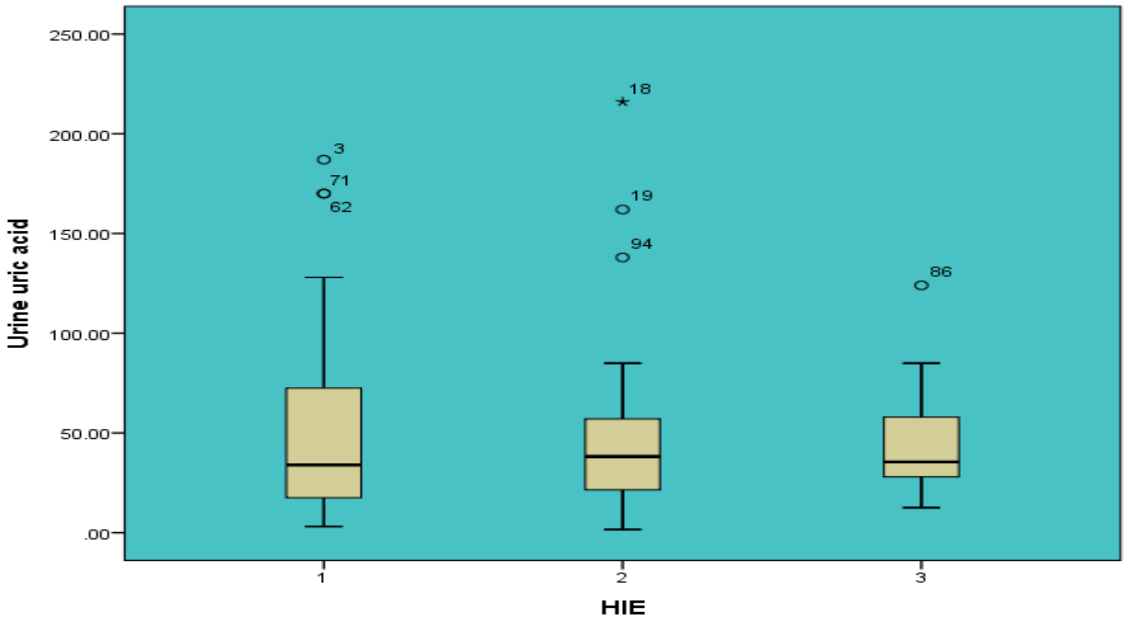


Figure 17: Comparative box plots of median urine creatinine across the HIE (N=one hundred and eight)

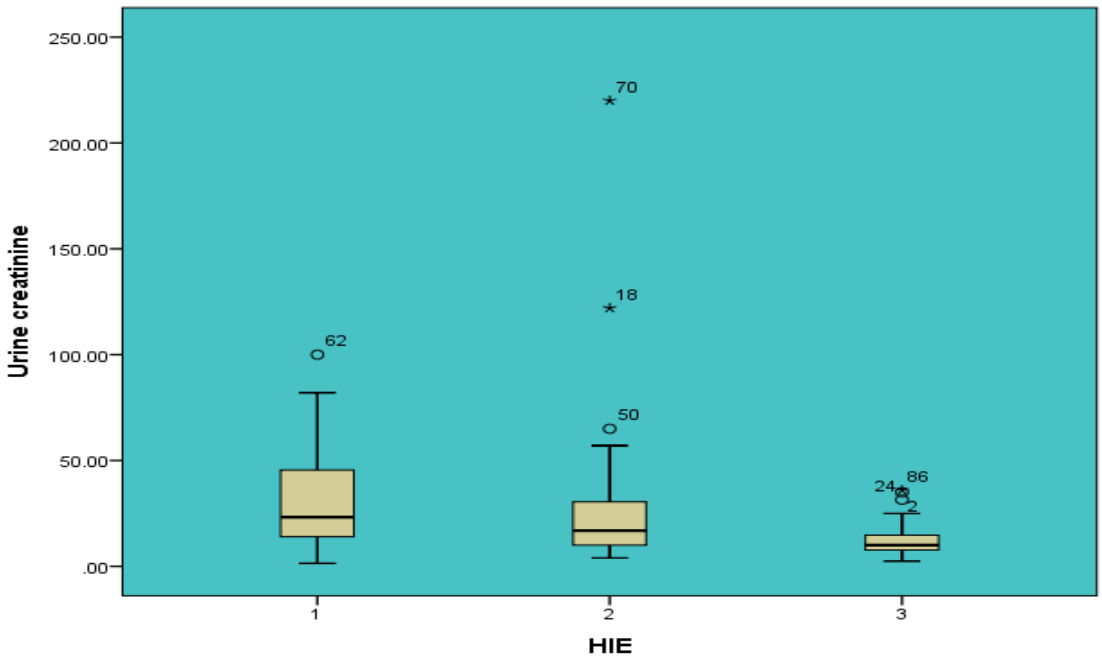
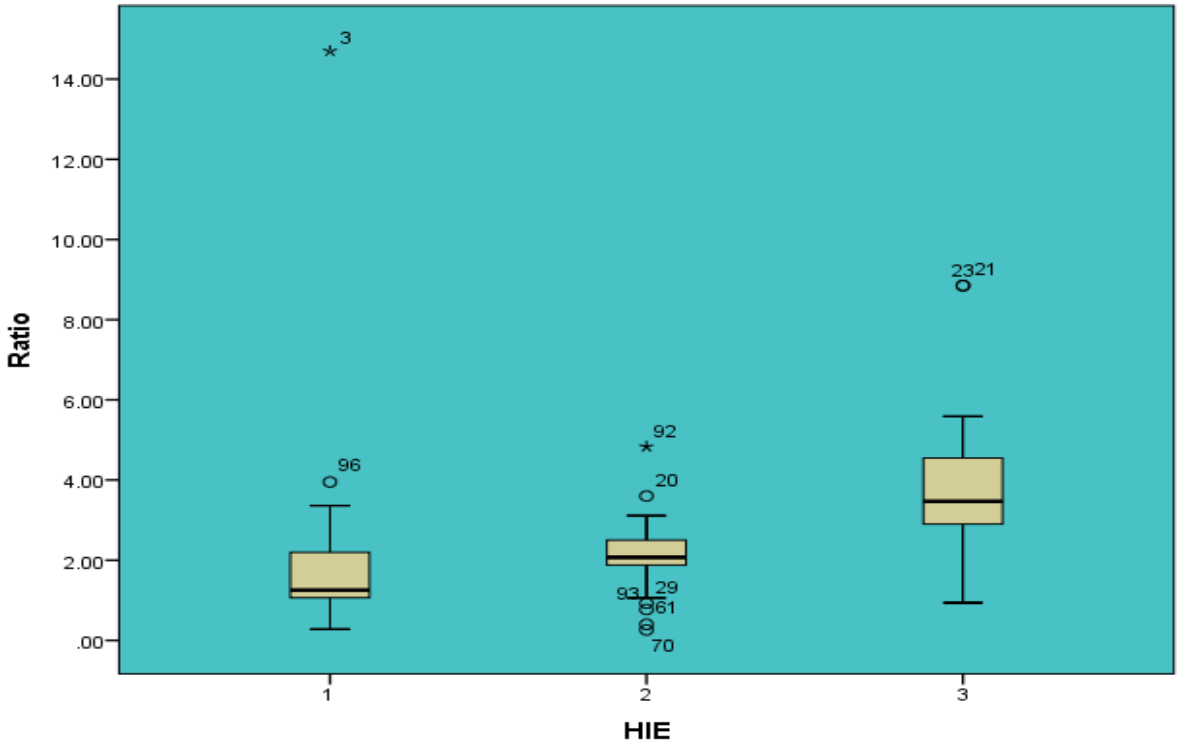


Figure 18: Comparative box plots of median ratio across the HIE (N=one hundred and eight)



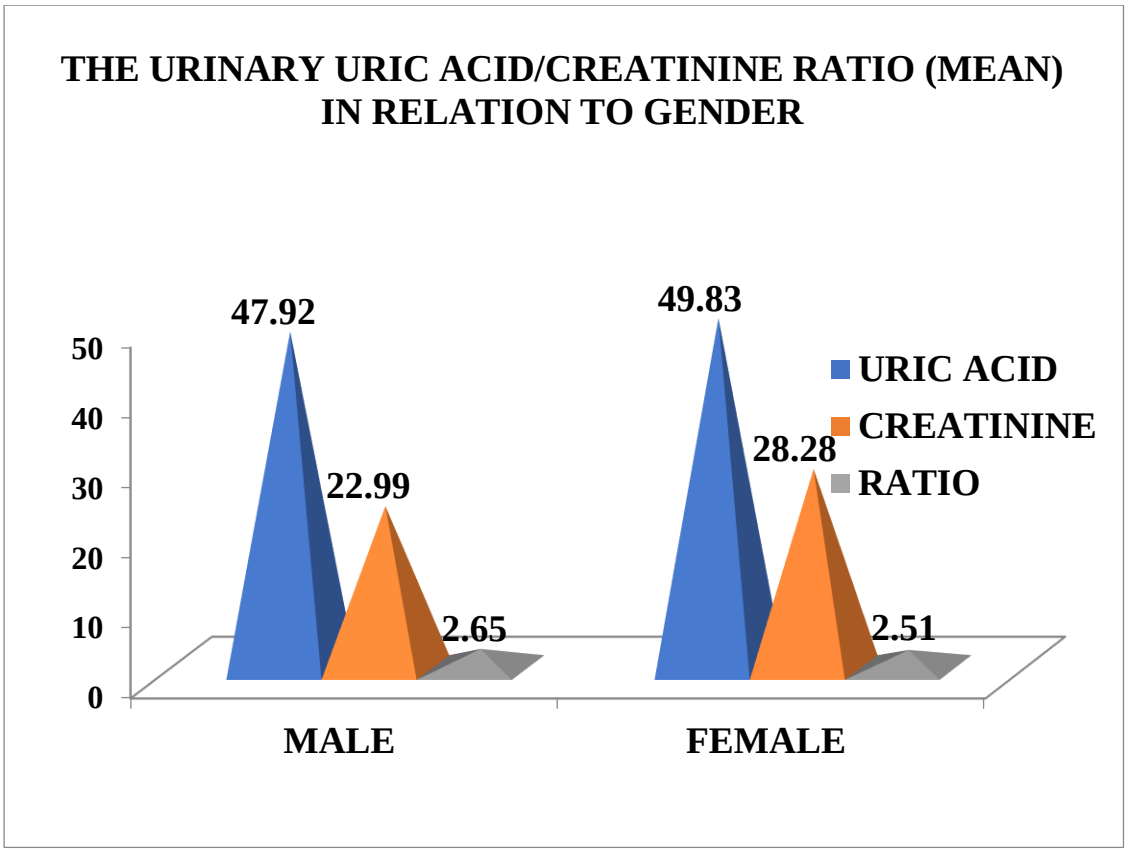


Figure 19: Bar diagram showing urinary uric acid and creatinine mean in relation to gender in study subjects a there is much significant difference.

Discussion

DISCUSSION

DISCUSSION:

Newborn asphyxia is one of the major causes of death worldwide and is defined as the failure to start breathing and maintain respiratory function in newborn infants. [21] Lawn *et al*, reported that the three most common causes of neonatal death are prematurity (28%), sepsis (26%) and asphyxia (23%) [21,22] and is on the most commonest cause of still birth (45.1%) in India.[23]

Prolonged hypoxia in newborn baby causes decrease in cardiac output which leads to the compromised cerebral blood flow and with combined hypoxic ischemic insult produces failure of ATP production with accumulation of ADP and AMP. Catabolism of these products leads to increase uric acid production with increased urinary excretion. Hypoxia and ischemia produced can cause damage to almost every tissue and organ of the body with common involvement of kidneys, brain, heart and lungs. [24]An estimated 1 million children who survive after experiencing asphyxia at birth are at higher risk of long-term morbidity such as cerebral palsy, mental retardation, and learning disabilities. [21,25]

Even though blood gas analysis remains the gold standard for establishing a diagnosis of newborn asphyxia, it is invasive and increases the risk of infection. There are various parameters have been used to predict or define perinatal asphyxia like cord blood pH, meconium stained amniotic fluid, APGAR score. Investigation of haematological parameters and basic biomarkers urine helps in the diagnosis of newborn asphyxia. Hence, the study was done with the purpose of assessing the possible association between Urinary uric acid and creatinine ratio in newborns with HIE. A total 108 people were included in the analysis.

Demographic profile

In the current study, the mean age at presentation was 13.67 ± 19.24 hours. 62 (57.40%) participants were male and remaining 46 (42.60%) were female. The mean duration of married life was 35.23 ± 20.75 months. 11 (10.20%) participants had 2nd degree consanguinity and 4 (3.70%) had 3rd degree consanguinity. The mean gestational age was 39.28 ± 1.05 in the study subjects. Mode of delivery was normal in 68 (63%), LSCS in 33 (30.60%), forceps assisted delivery in 4 (3.70) and vacuum assisted in 3 (2.80%). 5 (4.60%) subjects suffered from pre-eclampsia, 3 (2.80%) had present eclampsia. 18 (16.70%) children had meconium stained liquor and 3 (2.80) had PROM.

In the study by Akisu M *et al* [26] the mean gestational age was and mean birth weights were 39.2 ±1.4 weeks and the male female ratio was 15 (55.5%) /12 (44.5%) which was in accordance to the current study. The mode of delivery was vaginal for 17(62.9%), none of the subjects had Elective cesarean section and 10(37%) had Emergency cesarean section which was also in accordance to the present study.

In the study by Alkholy, U. M, *et al* [27], the mean gestational age was 39.2±1.24 (37–41). The male to female participants was 10 (40.0): 15 (60.0). Here, the number of females was more than the males; this was not in accordance to the current study. Mode of delivery was normal in only 12 (48.0) subjects and 13 (52.0) subjects underwent Cesarean section.

Choudhary, L, *et al*, in their study reported the Gestational age (weeks) to be 37.3 ± 1.82. The Male/Female ratio was 67(67%)/33(33%). The no. of vaginal deliveries was 61(61%) and number of LSCS deliveries was 39(39%). Vertex presentation was seen in 81(81%) children and Breech presentation in 10(10%) and other presentations in 9(9%).

The gestational age was 39.8 + 1.3 weeks in the study by Bader, D, *et al*.[28] only 28% had spontaneous delivery, none had elective Caesarian section, 39% had Emergency Caesarian section, and 33% had Forceps/vacuum delivery.

APGAR score:

In the present study, the APGAR score was analysed for only the 59 children as most of the children were born outside the current study setting. The mean APGAR score at 1 minute was 4.37 ± 1.11 in the study subjects, minimum level was 1 and maximum level was 7 (95% CI 4.08 to 4.66). The mean APGAR score 5 minutes was 6.25 ± 0.98 in the study subjects, minimum level was 3 and maximum level was 9 (95% CI 6 to 6.51). The mean APGAR score 10 minutes was 7.09 ± 1.1 in the study subjects, minimum level was 4 and maximum level was 9 (95% CI 6.79 to 7.39).

In the study by Akisu, M *et al* [26]APGAR score at 1 min was 2.5 (0-3) and at 5 min it was 3.0 (1-5). Alkholy, U. M, *et al*. [27] had the mean APGAR score of 2.0±1.7 (0–4) at 1 minute, 3.48±1.4 (1–7) at 5 minute and 5.15±1.4 (3–8) at 10 minute. In the study by Sreekrishna, Y., *et al*, [29] the mean APGAR score at 1 Min was 2.9±1.1, 5 Min was 5.1 ±1.0 and at 10 Min it was 7.1±0.8. The APGAR at 1st minute was 6.3 ± 1.3 and 8.3 ± 1.1 at 5th minute in the study by Bhongir, A. V, *et al*. [30]

Table 19 MEAN APGAR IN DIFFFERENT STUDIES

STUDY	1st MINUTE (MINIMUM, MAXIMUM)	5th M MINUTE (MINIMUM, MAXIMUM)INUTE	10th MINUTE MINUTE (MINIMUM, MAXIMUM)
OUR STUDY	4.37 ±1.11 (1-7)	6.25±0.98 (3-9)	7.09±1.1 (4-9)
Akisü, M *et al,*	2.5	3	NOT AVAILABLE
Alkholy.U.M,*et al,*	2±1.7	3.48±1.4	5.15±1.14
Sreekrishna, Y, *et al,*	2.9±1.1	5.1±1.0	7.1±0.8
Bhongir, A. V, *et al,*	6.9±1.3	8.3±1.1	NOT AVAILABLE

Mean APGAR is not coinciding with other studies because as it is observer dependent and it is dependent on many factors.

Anthropometric characteristics:

In the current study, the mean birth weight was 2.88 ± 0.43 in the study subjects, minimum weight was 1.34 kg and maximum weight was 3.86 kg (95% CI 2.80 to 2.96). The mean head circumference was 33.11 ± 0.96 in the study subjects, minimum level was 28.30 and maximum level was 35 (95% CI 32.93 to 33.29). The mean length was 48.41 ± 1.91 in the study subjects, minimum level was 38 and maximum level was 52 (95% CI 48.05 to 48.78). There are not many studies that have reported the anthropometric characteristics. However, mean birth weight has been reported by many studies. It was reported as 2.6±0.4 kg in the study by Jayaswal, A, *et al* [31], 3.1±0.4 (2.4–3.9) in the study by Alkholy, U. M, *et al,* [27] and 2.92 ± 0.67 in the study by Choudhary, L, *et al*.[32] and 2.6±0.4 in the study by Sreekrishna, Y, *et al*. [29]

HIE:

In the present study, 36 (33.30%) subjects had HIE 1, 40 (37%) had HIE 2 and 32 (29.60%) had HIE 3. In the study by Beken, S, *et al,* [33] 29(30.8%) subjects had HIE 1,

36(39.6%) had HIE 2 and 29(30.8%) had HIE 3. The number of asphyxiated babies in different stages of HIE were, stage-I 14(31%), stage-II 18(40%) and stage III 13 (29%) in the study by Vandana, V, *et al*.[24]

Urine uric acid median and Urine Uric acid creatinine ratio

In the present study, the mean urine uric acid was 48.7 ± 40.69 in the study subjects and the mean urine creatinine was 25.14 ± 28.69. The median Urine uric acid was 37.20(22.25, 61). The median urine creatinine was 15 units in the study subjects, with an interquartile range of 10 to 29.75. The median Urine Uric acid and creatinine ratio was 2.20, with an interquartile range of 1.51 to 3.18.In the study by Vandana, V, *et al,* [24] the mean Uric acid (mg/dl) was 34.1±14.8 in the asphyxiated group and 30.3±11.6 in the control group. The mean creatinine was 1.0±0.8 in asphyxiated group and 0.7±0.3 in the control group. Urinary uric acid and cratinine ratio was significantly higher (P<0.0001) in asphyxiated babies (3.02±1.26) compared to control group (0.84±0.56).

The urinary uric acid and cratinine ratio was 2.8±0.9 in the case group and 0.8±0.2 in the control group in the study by Sreekrishna, Y, *et al,* [29] The difference was statistically significant. This was similar to another study where the urinary uric acid and cratinine ratio was 2.58±1.09 in the case group and 0.86±0.17 in the control group in the study by Krishna, E., *et al,* [34]

Table 20 shows the urine uric acid and creatinine ratio comparison between different studies

	Present Study	Vandana,V, *et al*	Sreekrishna,Y, *et al*	Krishna, E, *et al*
Median urinary uric acid and creatinine ratio	2.2 IQR(1.51 -- 3.18	3.02±1.26	2.8±0.9	2.58±1.09

The above table shows the results of my study is similar with other two studies.

Association:

In the present study, among the HIE 1 subjects, the median urine uric acid was 34 (17.25, 75.75), it was 38.20 (20.75, 57.50) subjects with HIE 2 and it was 35.50 (28, 60.45) among the subjects with HIE 3. The difference in the urine uric acid across HIE was statistically not significant (P Value 0.864). Among the HIE 1 subjects, the median urine creatinine was 23.25 (14, 45.75), it was 16.90 (10, 31.25) subjects with HIE 2 and it was 10 (7.55, 15.05) among the subjects with HIE 3. The difference in the urine creatinine across HIE was statistically significant (P Value <0.001). Among the HIE 1 subjects, the median ratio was 1.26 (1.06, 2.20), it was 2.07 (1.87, 2.50) subjects with HIE 2 and it was 3.47 (2.85, 4.57) among the subjects with HIE 3. The difference in the ratio across HIE was statistically significant (P Value <0.001).

Akisü, M *et al* [26]found in their study, the urinary uric acid creatinine ratios were found to be higher in asphyxiated infants (2.1 I 0.83) when compared with the controls (0.72 f 03). P < 0001).Urinary uric acid and cratinine ratio were significantly higher in infants with severe HIE (3.15 to 0.81) when compared with infants with moderate HIE (2.13 to 0.36: P< 0.01) and those with mild HIE (1.43 i 0.29: P < 0.001). The value of the urinay uric acid creatinine ratios of the mild and moderate

HIE groups were also statistically different (P < 0.01). The results from the study by Banupriya, C., *et al*[35] the Spearman's correlation depicts that urine uric acid: creatinine ratio show a significant positive correlation with HIE staging and significant negative correlation with APGAR score.

Another study by Bader *et al*,[28] showed that Urinary uric acid and cratinine ratio was higher in the asphyxiated group when compared to controls. (2,06±1.12, vs. 0.64±0.48; P<0.001) which is also similar to our study. Our results are also supported by Chen *et al*.[36] who suggested that urinary ratio of UA to creatinine was significantly higher in both full term and preterm infants with perinatal asphyxia than in those without perinatal asphyxia.

Vandana *et al*[24] concluded in their study that theUrinary Urinary uric acid and cratinine ratio was significantly higher (P<0.0001) in asphyxiated babies (3.02±1.26) compared to control group (0.84±0.56). Mean values of urinary uric acid creatinine ratio in different stages of HIE showed increasing ratio with increasing stages of HIE (Table-III) with significantly higher ratios (P<0.0001) in stage II and III HIE (2.01±0.42 &4.24±0.79) compared to control group (0.84±0.56) and also compared to stage I HIE (1.23±0.52) (P<0.0001).

Summary

SUMMARY

An observational study was conducted by R.L. Jalappa Hospital and Research Centre, Department of Paediatrics attached to Sri Devaraj Urs Medical College, Kolar during January 2017 to May 2018. A total of 108 asphyxiated babies were included during the study period.

Babies with hypoxic ischemic encephalopathy as per sarnath and sarnath staging were included in the study. Detailed maternal history, birth events, APGAR score, sex of the baby and anthropometry of the baby were recorded on the proforma. Thorough clinical and neurological examination was done for all the neonates included in the study.

The babies who has suffered asphyxia and as per standard staging system (sarnat and sarnat) which fit in to the criteria of HYPOXIC ISCHEMIC ENCEPHALOPATHY will be categorized in to stage1, stage 2 and stage 3 and included in the study.

The asphyxiated neonates were monitored for all components of sarnat and sarnat staging in the immediate neonatal period in the NICU. Grading system used to grade the severity of HIE was SARNAT and SARNAT staging 1976. The urinary samples were collected as early as possible baby diagnosed clinically to have HIE and all urine samples were collected by staff nurse in sterilized disposable urine bag and analyzed in hospital laboratory for Urinary uric acid to creatinine ratio. Urinary uric acid was estimated by auto analyzer by spectrophotometric uricase method. Urinary creatinine was estimated in same above instrument by using sarcosine oxidase method. Data was entered in MS excel and analyzed using IBM SPSS 22 VERSION software. Qualitative data is presented in the form of proportions and pie diagrams, bar charts used to represent graphically. Quantitative data is presented as mean, standard

deviation. Descriptive analysis was carried out by mean and standard deviation for quantitative variables, frequency and proportion for categorical variables. Data was also represented using appropriate diagrams like bar diagram, pie diagram and box plots.

All Quantitative variables were checked for normal distribution within each category of explanatory variable by using visual inspection of histograms and normality Q-Q plots. Shapiro- wilk test was also conducted to assess normal distribution. Shapiro wilk test $p<0.05$ was considered as significant.

For none normally distributed Quantitative parameters, Medians and Interquartile range (IQR) were compared across the study groups using Kruskal Wallis test (> 2 groups).

$P< 0.05$ was considered statistically significant. IBM SPSS version 22 was used for statistical analysis.[1] Among the HIE 1 subjects, the median urine uric acid was 34 (17.25, 75.75), it was 38.20 (20.75, 57.50) subjects with HIE 2 and it was 35.50 (28, 60.45) among the subjects with HIE 3. The difference in the urine uric acid across HIE was statistically not significant (P Value 0.864).

Among the HIE 1 subjects, the median urine creatinine was 23.25 (14, 45.75), it was 16.90 (10, 31.25) subjects with HIE 2 and it was 10 (7.55, 15.05) among the subjects with HIE 3. The difference in the urine creatinine across HIE was statistically significant (P Value <0.001). Among the HIE 1 subjects, the median ratio was 1.26 (1.06, 2.20), it was 2.07 (1.87, 2.50) subjects with HIE 2 and it was 3.47 (2.85, 4.57) among the subjects with HIE 3. The difference in the ratio across HIE was statistically significant (P Value <0.001). (Table 23 & Figure 2,3and 4)

Conclusion

CONCLUSION

- A total of 108 subjects were included in the study. The mean age at presentation was 13.67 ± 19.24 hours in the study subjects, ranged between 0.27 to 144 hours and 62 (57.40%) subjects were male and 46 (42.60%) were females.

- The mean gestational age was 39.41± 1.00 in the study subjects. Mode of delivery was normal in 68 (63%), LSCS in 33 (30.60%), forceps assisted delivery in 4 (3.70) and vacuum assisted in 3 (2.80%). Only 1 (0.90%) subjects had postdated and 107 (99.10) had term pregnancy. 5 (4.60%) subjects suffered from pre-eclampsia. Among the study subjects, 3 (2.80%) had present eclampsia. Among the study subjects, 18 (16.70%) subjects had present meconium stained liquor and 3 (2.80) had prom.

- APGAR score was analyzed for only the 59 children as most of the children were born outside the current study setting**.** The mean APGAR score at 1 minute was 4.37 ± 1.11 in the study subjects, minimum level was 1 and maximum level was 7 (95% CI 4.08 to 4.66). The mean APGAR score 5 minutes was 6.25 ± 0.98 in the study subjects, minimum level was 3 and maximum level was 9 (95% CI 6 to 6.51). The mean APGAR score 10 minutes was 7.09 ± 1.1 in the study subjects, minimum level was 4 and maximum level was 9 (95% CI 6.79 to 7.39).

- The mean birth weight was 2.88 ± 0.43 in the study subjects, minimum weight was 1.34 kg and maximum weight was 3.86 kg (95% CI 2.80 to 2.96). The mean head circumference was 33.11 ± 0.96 in the study subjects, minimum level was 28.30 and maximum level was 35 (95% CI 32.93 to 33.29). The mean length was 48.41 ± 1.91 in the studysubjects, minimum level was 38 and maximum level was 52 (95% CI 48.05 to 48.78).

- Among the study subjects, 98 (90.70%) had pink color, 4 (3.70%) had pale color and 6 (5.60%) had cyanosis color. Among the study subjects, 59 (54.60%) had good cry, 9

(8.30%) had moderate and 40 (37%) had poor. Among the study subjects, 57 (52.80%) had good suck, 9 (8.30%) had moderate and 42 (38.90%) had poor. Among the study subjects, 57 (52.80%) had good tone, 9 (8.30%) had moderate and 42 (38.90%) had poor. Among the study subjects, 58 (53.70%) had good activity, 9 (8.30%) had moderate and 41 (38.00%) had poor.

- The mean heart rate was 149.58 ± 24.05, 6 (5.60%) subjects had cyanosis. 106 (98.10%) subjects had present peripheral pules and 2 (1.90%) had feeble pulse. The mean SPO_2 was 89.02 ± 12.05 in the studysubjects.

- Among the study subjects, 36 (33.30%) had HIE 1, 40 (37%) had HIE 2 and 32 (29.60%) had HIE 3.

- The mean urine uric acid was 48.7 ± 40.69 in the study subjects and the mean urine creatinine was 25.14 ± 28.69.

- The median Urine uric acid median was 37.20 (22.25, 61). The median urine creatinine was 15 units in the study subjects, with an interquartile range of 10 to 29.75. The median Urine uric acid and creatinine ratio was 2.20, with an interquartile range of 1.51 to 3.18.

- Among the HIE 1 subjects, the median urine uric acid was 34 (17.25, 75.75), it was 38.20 (20.75, 57.50) subjects with HIE 2 and it was 35.50 (28, 60.45) among the subjects with HIE 3. The difference in the urine uric acid across HIE was statistically not significant (p 0.864). Among the HIE 1 subjects, the median urine creatinine was 23.25 (14, 45.75), it was 16.90 (10, 31.25) subjects with HIE 2 and it was 10 (7.55, 15.05) among the subjects with HIE 3. The difference in the urine creatinine across HIE was statistically significant ($p<0.001$). Among the HIE 1 subjects, the median ratio was 1.26 (1.06, 2.20), it was 2.07 (1.87, 2.50) subjects with HIE 2 and it was 3.47 (2.85, 4.57) among the subjects with HIE 3. The difference in the ratio across HIE was statistically significant ($p <0.001$).

LIMITATIONS:

1. Due to limited sample size of the studysubjects, we could not establish the independent association between the grade of asphyxia and urinary uric acid creatinine ratio, after controlling for the effect of other potential confounding variables.
2. Non availability of data on APGAR scores for close to half of the children, as they were referred from outside hospitals was another key limitation.
3. The generalizability of the study findings is limited, as the study was conducted in a single tertiary care institution, with limited catchment area.

RECOMMENDATIONS:

1. There is a need to conduct further large scale prospective studies on the association between birth asphyxia and urinary uric acid and creatinine ratio and it's impact on further course of the disease and ultimate recovery status.

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Annexures

PROFORMA

CORRELATION OF URINARY URIC ACID, CREATININE RATIO WITH THE SEVERITY OF HYPOXIC ISCHEMIC ENCEPHALOPATHY

Name of Mother : Age: Hospital number:
Name of Father : Age:
Address : Sex: Age in hours:
Date of Birth: Time of Birth:
Date of NICU Admission : Time of NICU Admission:

ANTENATAL EVENTS:

Married life: Consanguinity: Gestational Age:

Preeclampsia: Eclampsia: Bleeding Diathesis:

Hypertension: Drugs: Others:

PERIPARTUM EVENTS:

Antepartum Hemorrhage: CTG:

Foetal Distress: Mode Of Presentation:

Place Of Birth: APGAR Score: 1" - /10 5"- /10 10" - /10

Birth Weight: MSAF: PROM:

Resuscitation:

Events: Requires 02: Outcome :

IPPV: Routine Neonatal Care:

Intubation: Post Resuscitation Care:

Drugs:

Chorionamniotis:

History Of Seizures:

Date of Convulsion: Time of Convulsion:

Duration of convulsion: Episode of Convulsion:

Associated Autonomic Changes:

General Examination-

Color: Tone- Activity-
Pulse Rate : / min Respiratory Rate- / min Pallor-
Cyanosis:
Dysmorphic features:
Anthroprometry-Birthweight:
Head Circumference: Length:
AGA: SGA: LGA-Ponderal Index-

Systemic Examination:
Central Nervous System:
Alert- Cranial Nerve :
Motor: Reflex:
Seizure Profile: Age at Onset Of Seizures (in hrs)-
Duration Of Seizures: <5 min- 5min-30 min- >30min-
Type of Seizures:
Focal Clonic: Focal Tonic: Generalised Tonic:
Myoclonic: Spasms: Motor Automatism:
Response To Drugs: Time Taken To Response:
Immediate(<5mins)
Delayed(5mins-30 mins)
Status Epilepticus(>30 mins)
Associated Autonomic Changes: Present Absent
Changes Present:
Heart Rate: / min Respiratory Rate: /min Spo2-
Cardio Vascular System:
Heart Rate: /min Peripheral pulses: Spo2:
Capillary Filling time: sec Murmurs-
Respiratory System:
Respiratory Rate: / min Chest Retractions- Grunting-
Air Entry:
PERABDOMEN:
Umbilical Arteries / Veins: Patency Of Anus-
Organomegaly: Liver-Spleen- Renal-
Clinical Diagnosis:
Extra Cranial: Intra Cranial:
Birth Injury:
HIE: I: II: III:
Dysmorphology:

PATIENT INFORMATION SHEET

Title of the study:

CORRELATION OF URINARY URIC ACID, CREATININE RATIO WITH THE SEVERITY OF HYPOXIC ISCHEMIC ENCEPHALOPATHY

- **Purpose of the Research:**
- Due to some limitations, the APGAR score cannot be used as a useful tool for the evaluation of newborns.
- There are different scoring systems for assessing neonatal mortality.These scoring systems contain many items, and sometimes checking takes hours; as a result, they can be very time-consuming in some situations.
- But APGAR score alone does not predict neurologic outcome like cerebral palsy and as it is influenced by various factors like immaturity, fetal malformations, maternal medications and infection. Whereas APGAR score and umbilical artery blood pH both predict the neonatal mortality in term and preterm infants.Indicators such as pH, lactates and base deficits subside with the establishment of respiration, and moreover these techniques are costly and sophisticated
- Studies have reported higher urinary Urinary uricacid and cratinine ratio ratio in preterm and term infants with perinatal asphyxia than in normal infants. Studies have compared urinary Urinary uricacid and cratinine ratio ratio with APGAR and some with Cord blood Ph but none of them compared with both APGAR score and cord blood PH, saranat staning. Hence, this study was conducted to assess the urinary uricacid and cratinine ratio(Urinary uricacid and cratinine ratio) in relation to APGAR score and cord blood gas analysis, HIE staging
- Accurate assessment of late neurological squeal has failed by implementing strategies such as fetal heart monitoring[3], APGAR score, while analysis of xanthine, hypoxanthine, neuron specific enolase,brain-specific creatinine kinase and inflammatory cytokines (tumor necrosis factor-alpha, interleukin-1-beta, interleukin-8, interleukin-6) are time consuming, costly and not routinely available for clinical care

Procedures and Protocols

1.Detailed maternal history, birth events, APGAR score, sex of the baby and weight of the baby were recorded on the recoded Performa.

2 Gestational age is assessed by New Ballard scoring system.

3. Thorough clinical and neurological examination was done for all the neonates included in the study.

4.The babies who has suffered asphyxia and as per standard staging system(sarnat and sarnat) which fit in to the criteria of HYPOXIC ISCHEMIC ENCEPHALOPATHY will be categorized in to stage1 to 3 and included in to the study group

3. The asphyxiated neonates (case group) were monitored for seizures, hypotonia and HIE in the immediate neonatal period in the ICU. Grading system used to grade the severity of HIE was SARNAT and SARNAT staging 1976.

Stage	Stage1(MILD)	Stage2(Moderate)	Stage3(Severe)
Level of consciousness	Hype alert, irritable	Lethargic or obtunded	Stuporous, comatose
Neuromuscular control:	Uninhibited, over reactive	Diminished spontaneous movement	Diminished or absent spontaneous movement
Muscle tone	Normal	Mild hypotonia	Flaccid
Posture	Mild distal flexion	Strong distal flexion	Intermittent decerebration
Stretch reflexes	Overactive	Overactive, disinhibited	Decreased or absent
Segmental myoclonus	Present or absent	Present	Absent
Suck	Uninhibited, over reactive	Diminished spontaneous movement	Diminished or absent spontaneous movement
Moro	Normal	Mild hypotonia	Flaccid
Oculovestibular	Normal	Overactive	Weak or absent
Tonic neck	Slight	Strong	Absent
Autonomic function:	Generalized sympathetic	Generalized parasympathetic	Both systems Depressed
Pupils	Mydriasis	Miosis	Midposition, often unequal; poor light reflex

Respirations	Spontaneous	Spontaneous; occasional apnea	Periodic; apnea
Heart rate	Tachycardia	Bradycardia	Variable
Seizures	None	Common focal or multifocal (6-24 hours of age)	Uncommon (excluding decerebration)
Electroencephalographic Findings	Normal (awake)	Early: generalized low voltage, slowing (continuous delta and theta) Later: periodic pattern (awake); seizures focal or multifocal; 1.0-1.5 Hz spike and wave	Early: periodic pattern with isopotential phases Later: totally Isopotential
Duration of symptoms	<24 hours	2-14 days	Hours to weeks
Outcome	About 100% normal	80% normal; abnormal if symptoms more than 5-7 days	About 50% die; remainder with severe sequelae

Reimbursements: You will not be given money or gifts to take part in this research.

Confidentiality: We will not be sharing the identity of the participant. The information we collect from you will be kept confidential and only the researches involved in this project will have access to it.

Right to Refuse or Withdraw: You do not have to take part in this research if you do not wish to do so and you can refuse to participate.

Who to Contact: If you have any questions you may ask us now or later, even after the study has started you may contact the following persons.

For more Information:

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Post Graduate in Pediatrics

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CONSENT FORM

INFORMED CONSENT FORM

I, Mr/Mrs have been explained in a language I can understand, that I will be included in a study which correlation of urinary uric acid, creatinine ratio with the severity of hypoxic ischemic encephalopathy

.

I have been explained that my clinical finding, investigations, lab values will be assessed and documented for the study purpose.

I have been explained that my participation in this study is entirely voluntary and I can withdraw from the study anytime and this will not affect my relation with my doctor or the treatment for my ailment.

I have understood that all my details found during the study are kept confidential and while publishing or sharing of the findings, my details will be masked.

I, in my sound mind give full consent to be added in the part of this study.

Signature of the patient:

Name:

Signature of the witness:

Name:

Date: Place:

Institutional Ethical Clearance for PG Students

SRI DEVARAJ URS MEDICAL COLLEGE
TAMAKA, KOLAR – 563101

ETHICS COMMITTEE CERTIFICATE

This is to certify that the ethics committee of Sri Devaraj Urs Medical College, Kolar in its meeting conducted on **16-12-2016** has unanimously approved the synopsis for the dissertation entitled **"Correlation of urinary uric acid creatinine ratio with the severity of hypoxic ischemic encephalopathy"** to be submitted to Sri Devaraj Urs Academy of Higher Education and Research, Kolar, Karnataka, by **Dr.Kota Venkata Suman**, Postgraduate student in the department of **MD Paediatrics** at Sri Devaraj Urs Medical College, Kolar

Member Secretary
Institutional Ethics Committee
SDUMC, Tamaka Kolar

Member Secretary
Institutional Ethics Committee
Sri Devaraj Urs Medical College
Tamaka, Kolar.

Chairman
Institutional Ethics Committee
SDUMC, Tamaka Kolar

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Sri Devaraj Urs Medical College.
Tamaka, Kolar

Date : 19-12-2016
Place: Kolar

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Dr. N. C. Jain
Scientist- G & Head (HRD)

No.3/2/Dec. 2016/PG-Thesis-HRD (19)
Dated: 22.03.2017

To,
Dr. Kota Venkata Suman
Deptt. of Paediatrics,
Sri Devaraj Urs Medical College,
Tamaka, Kolar-563101
drsuman42@gmail.com

Dear Dr. Kota Venkata Suman,

This is with reference to your application seeking financial assistance from the ICMR for MD/MS/DM/MCh dissertation thesis entitled "**Correlation of urinary uric acid, creatinine ratio with the severity of hypoxic ischemic encephalopathy**".

I am glad to inform you that Director General, ICMR, based on the recommendation of Expert Committee, has sanctioned a sum of **₹. 50, 000/- (Fifty thousand only)** to you for providing an electronic and hard copy of your dissertation thesis to the ICMR. Mandatory requirement to avail this opportunity is to provide us with an undertaking duly forwarded through the guide, to the undersigned, enabling us to release the grant.

This is to inform you that ₹. 50,000/- will be disbursed to you in two installments. Initial amount of ₹. 35,000/- after receipt of the undertaking as per the guidelines and remaining amount of ₹. 15,000/- on receipt of the electronic copy, hard copy and summary of work done of your dissertation thesis duly approved by the University/ Institute along with one publication in an indexed Journal.

The amount will be released after submitting undertaking as well as the mandate form (icmr.nic.in) for receiving e-payments along with a photocopy of a cancelled cheque for purpose of verification of the concerned bank account where money is to be remitted.

With best wishes,

Yours faithfully,

(N. C. Jain)
011-26589258
drncejain@gmail.com

Copy to: Dr. Beeregowda Y.C., Professor, Deptt. of Paediatrics, Sri Devaraj Urs Medical College, Tamaka, Kolar-563101.

KEY TO MASTER CHART SHEET

A) Serial Number -SN

B) Hospital number-HN

C) Name-N

D) Age-A

- **H– Hours**
- **M- Minutes**
- **D-Days**

E) Sex

- **M- Male**
- **F- Female**

F) Duration of married life

- **Y- Years**
- **M- Months**

G) Consanguinity

- **Y- Yes**
- **N-No**
- **1-First degree**
- **2-Second degree**
- **3-Third degree**

H) Gestational age

- **W- Weeks**
- **D- Days**

I) Mode of delivery

- **NVD- Normal vaginal delivery**
- **FAD-Forceps assisted delivery**
- **VAD-Vaccum assisted delivery**
- **LSCS-Lower segment caesarian section**

J) Term/Post term/Post dated

- **T-Term**
- **PT- Post term**
- **PD-Post dated**

K) Preeclamsia

- **Y- Yes**
- **N- No**

L) Eclamsia

- **Y- Yes**
- **N- No**

M) Bleeding diathesis

- **Y- Yes**
- **N- No**

N) Ante partum haemorrhage

- **Y-Yes**
- **N-No**

O) Meconium stained liquor

- **Y-Yes**
- **N-No**

P) PROM/PPROM

- **PROM-Premature rupture of membranes**
- **PPROM-Preterm premature rupture of membranes**
- **Y-Yes**
- **N-No**

Q) Chorioamnitis

- **Y-Yes**
- **N-No**

R) Others

- **RS-Renal stones**
- **M-Month**
- **MOH-Mild oligohydromnios**
- **F-Fever**
- **CRP-C-reactive protein**
- **CAN-Cord around neck**
- **IDM-Infant of diabetic mother**
- **HM-Hypothyroid mother**
- **RU-Ruptured uterus**
- **H/O F –History of fever**

S) APGAR SCORE 1 Min

- **APGAR- Appearance, Pulse, Grimace,Activity, Respiration**
- **NK-Not known**
- **MIN-Minutes**

T) APGAR SCORE 5 Min

U) APGAR SCORE 10Min

V) Birth weight in kgs

- **Kgs-Kilograms**

W) Head circumference

X) Length

Y) Colour

- **PI-Pink**
- **PA-Pale**
- **C-Cyanosis**

Z) Cry

- **P-Poor**
- **M-Moderate**
- **G-Good**

AA) Suck

- **P-Poor**
- **M-Moderate**
- **G-Good**

BB) Tone

- **P-Poor**
- **M-Moderate**
- **G-Good**

CC) Activity

- **P-Poor**
- **M-Moderate**
- **G-Good**

DD) Heart rate

EE) Respiratory rate

- **G-Gaping**
- **MV-Minute ventilation**

FF) Cyanosis

- **N-Nil**
- **P-Present**

GG) Duration of seizures

- **N-Nil**
- **M-Minutes**
- **S-Status**

HH) Peripheral pulses

- **P-Present**
- **A-Absent**
- **F-Feeble**

II) Spo2 baseline

- **Spo2-Saturation of peripheral oxygen**

JJ) HIE Stage

- **HIE- Hypoxic ischaemic encephalopathy**

KK) Urine uric acid

LL) Urine creatinine

MM) Urine uric acid /urine creatinine ratio

S.No	UH ID NO	Name	AGE OF PRESENTATI	GENDER	Duration of marriec	Consanguinity	Gestational age in V	mode of delivey	T/POST TERM / POS	Pre Eclampsia	Eclampsia	bleeding Diathesis	Antepanrtum Haen	meconium stained li	PROM	Chorioamnitis	others	Apgar score 1	Apgar score5	Apgar 10	Birth weight in kg	Head circumferance	length	Color	CRY	SUCK	Tone	Activity	heart rate	RR	cyanosis	Duration of seizures	peripheral pulses	Spo2 @ baseline	HIE	URINE URIC ACID	urine creatinine	ratio
1	423266	B/O RUKSANA BEGUM	30M	F	1Y6M	2	38W 3D	NVD	T	N	N	N	N	N	N	N		5	6	7	3.28	34	49	PI	P	P	P	P	160	72	N	2M	P	85	3	34	16	2.12
2	445401	b/omala	4H	F	1Y6M	N	40W 2D	NVD	T	N	N	N	N	N	N	N		3	6	7	3.34	33	48	PI	P	P	P	P	120	48	N	2M	P	79	3	33	35	0.94
3	469588	b/o sagarika	1H	F	2Y	N	41W4D	FAD	T	N	N	N	N	N	N	N		3	7	8	2.56	33	48	PI	M	M	M	M	165	72	N	N	P	96	1	187	12.7	14.7
4	465716	b/o vanja	3H	F	3Y	N	40W	NVD	T	N	Y	N	N	N	N	N		3	6	6	3.4	33	49	PA	P	P	P	P	220	74	Y	N	P	50	3	28.4	7.4	3.83
5	410064	b/o manjula	1hr	M	4Y	3	37W	LSCS	T	Y	N	N	N	N	N	N		5	8	8	1.34	28.3	48	PI	G	G	G	G	135	45	N	1M	P	95	2	44.6	16.8	2.65
6	432273	b/o tabark	24H	M	1Y	N	39W 2D	NVD	T	N	N	N	N	N	N	N		3	6	6	2.8	34.5	48	PI	P	P	P	P	112	G	N	N	P	82	3	40.3	20.3	1.98
7	410155	b/o manjula	1H30M	M	1Y	N	40W3D	LSCS	T	N	N	N	N	N	N	N		3	7	7	2.74	33	50	PI	G	G	G	G	130	42	N	N	P	96	1	43.2	20	2.16
8	475436	b/o sunitha	43H	F	2Y	N	40W	NVD	T	N	N	N	N	N	N	N		6	8	8	3.2	32	48	PI	M	M	M	M	154	68	N	N	P	74	1	3.1	1.4	2.21
9	472769	b/o madavi	68H	F	1Y	N	40W6D	LSCS	T	N	N	N	N	N	N	N	RS AT 7M	6	8	8	2.8	32	48	PI	G	G	G	G	150	45	N	N	P	96	1	5.7	3.1	1.84
10	447649	b/o munirathna	24H	M	1Y	N	39W6D	NVD	T	N	N	N	N	N	N	N	MAH	5	6	6	2.7	31.5	45	PI	M	M	M	M	148	48	N	2M	P	96	2	80.4	40.2	1.99
11	479936	b/o lakshmidevi	15H	M	1Y6M	N	38W	LSCS	T	N	N	N	N	N	N	N		7	9	9	3	34	52	PI	G	G	G	G	140	40	N	N	P	96	1	13.6	11	1.23
12	423266	B/O RUKSANA BEGUM	6H	F	1Y 6M	N	38W3D	LSCS	T	N	N	N	N	N	N	N		5	6	7	3.18	34	49	PI	G	G	G	G	144	48	N	1M	P	80	2	31.36	16	1.96
13	416535	b/o sangeetha	66H	M	2Y	N	37W6D	LSCS	T	N	N	N	N	N	N	N		4	6	7	2.62	34	48	PI	M	M	M	M	150	42	N	1M	P	96	2	40	20	2
14	416889	b/o shoba	10H	M	4Y	2	38W6D	LSCS	T	N	N	N	N	N	N	N		5	6	7	3.2	34	48	PI	G	G	G	G	140	46	N	N	P	98	1	55	23	2.3
15	551314	b/o amrutha	30H	M	3Y	N	40W	NVD	T	N	N	N	N	N	N	N	N	NK	NK	NK	3	33	50	PI	G	G	G	G	140	56	N	1M	P	96	2	13	6.5	2
16	450343	b/o sakamma	17H	F	3Y	N	39W	NVD	T	N	N	N	N	N	N	N	F AND CRP +VE	5	6	6	2.5	33	47.5	PI	G	G	G	G	165	60	N	1M	P	85	2	85	29	2.93
17	445450	b/o mamatha	6H	M	13Y	3	40W	NVD	T	N	N	N	N	N	N	N		5	6	6	3.2	34	48	PI	G	G	G	G	122	40	N	1M	P	96	2	51	20	2.5
18	409417	b/o shashikala	16H	M	3Y	N	37W3D	NVD	T	N	N	N	N	N	N	N		5	6	6	2.6	32	49	PI	G	G	G	G	126	69	N	1M	P	97	2	216	122	1.77
19	445984	b/o devani	30M	M	10Y	2	39W5D	LSCS	T	N	N	N	N	N	N	N		5	6	6	3.04	33	52	PI	G	G	G	G	122	42	N	1M	P	96	2	162	52	3.11
20	429187	b/o shilaja	4H	F	1Y	2	41W1D	NVD	T	N	N	N	N	N	N	N		5	6	6	3.6	34.5	52	PI	M	M	M	M	148	64	N	1M	P	80	2	36	10	3.6
21	486224	b/o mallika	20M	M	2Y	N	39W	NVD	T	N	N	N	N	N	N	N		4	5	6	2.32	34	48	C	P	P	P	P	154	65	N	N	P	96	3	85	9.6	8.85
22	486224	b/o mallika	16M	M	4Y	N	39W	NVD	T	N	N	N	N	N	N	N		4	5	6	2.32	34	49	C	P	P	P	P	154	62	N	1M	P	96	2	39	14	2.78
23	488521	b/o vani	80M	F	1Y 6M	N	38W	VAD	T	N	Y	N	N	N	N	N		3	6	6	3.5	34	48	PI	P	P	P	P	164	66	N	N	P	50	3	85	9.6	8.85
24	488521	b/o vani	80M	F	1Y 6M	N	38W	VAD	T	N	N	N	N	N	N	N		3	6	6	3.5	34	49	PI	P	P	P	P	164	66	N	N	P	50	3	85	31.5	2.6
25	612397	b/o shahistha	30M	F	4Y		40W	NVD	T	Y	N	N	N	Y	N	N	N	NK	NK	NK	2.5	33	48	PI	P	P	P	P	166	70	N	1M	P	90	2	14.3	7.7	1.85
26	505120	b/o rekha	30M	M	1Y6M	2	38W 3D	NVD	T	N	N	N	N	N	N	N		5	6	7	3.28	34	49	PI	G	G	G	G	160	72	N	N	P	96	1	55	54	1.08
27	505119	b/o usha	4H	F	2Y	N	40W 2D	LSCS	T	N	N	N	N	N	N	N		5	6	7	3.34	33	48	PI	G	G	G	G	120	48	N	N	P	97	1	48	46	1.04
28	482928	b/o rajeshwari	1H	M	3Y	N	41W4D	NVD	T	N	N	N	N	N	N	N		5	7	8	2.56	33	48	PI	G	G	G	G	165	72	N	N	P	96	1	38	42	0.9
29	470488	b/o shailaja	5H	F	1Y	2	36W2D	LSCS	T	Y	N	N	N	N	N	N		5	6	7	1.54	30	40	PA	M	M	M	M	160	74	N	1M	P	98	2	39	45	0.928
30	440533	b/o bibi hezara	1H	F	4Y	3	37W	LSCS	T	Y	N	N	N	N	N	N		6	8	8	2.85	33	48	PI	G	G	G	G	135	45	N	N	P	97	1	29	39	0.74
31	438072	b/o asfia sulthana	30M	M	5Y	N	38W 5D	VAD	T	Y	N	N	N	N	N	N		4	6	8	2.8	32	48	PI	G	G	G	G	150	60	N	N	P	92	1	26	24	1.08
32	418954	b/o sudharani	30M	F	4Y	N	39W4D	NVD	T	N	N	N	N	N	N	N	CAN	4	5	6	3.22	33	48	PI	G	G	G	G	179	64	N	N	P	98	1	28	26	1.07
33	392948	b/o sushelamma	6H	M	4Y	N	40W6D	NVD	T	N	N	N	N	N	N	N		6	8	8	2.8	33	48	PI	G	G	G	G	150	45	N	N	P	96	1	19	28	0.64
34	393299	b/o ambika	30M	M	1Y3M	N	38W3D	NVD	T	N	N	N	N	N	N	N		6	7	9	2.98	34	49	PI	G	G	G	G	132	52	N	N	P	95	1	18	16	1.12
35	616408	b/o amrutha	4H	M	4Y	N	39W	NVD	T	N	N	N	N	N	N	N	N	NK	NK	NK	2.8	33	48	PI	G	G	G	G	147	50	N	1M	P	94	2	23	9	2.5
36	375646	b/o anjum kouser	10H	F	1Y6M	N	38W3D	LSCS	T	N	N	N	N	N	N	N		6	6	7	3.18	34	49	PI	G	G	G	G	144	48	N	N	P	97	1	30	54	0.5
37	507340	b/o nasreen k	31H	M	1Y	N	40W 4D	NVD	T	N	N	N	N	N	N	N		5	7	9	3	34	48	PI	G	G	G	G	145	47	N	N	P	96	1	39	69	0.56
38	506377	b/o renuka	2H	M	6Y	N	39W	NVD	T	N	N	N	N	N	N	N		5	7	8	2.62	33	46	PI	G	G	G	G	156	48	N	1M	P	92	2	48	19	2.52
39	504482	b/o mamatha	16H	F	4Y	N	40W3D	LSCS	T	N	N	N	N	N	N	N		3	6	8	3.34	33	48	PI	P	P	P	P	169	46	N	3M	P	94	3	38	12	3.1
40	500211	b/o nagarathnamma	3H	F	1Y	N	39W6D	NVD	T	N	N	N	N	N	N	N	F	4	6	6	3	33	49	PI	M	M	M	M	124	32	N	1M	P	96	2	82	40	2.05
41	499718	b/o geetha	6H	M	2Y	2	40WW	NVD	T	N	N	N	N	N	N	N		4	6	8	2.5	32	46	PI	M	M	M	M	156	48	N	1M	P	94	2	31	15	2.06
42	480924	b/o roopa	30M	M	4Y	N	39W6D	NVD	T	N	N	N	N	N	N	N		4	6	8	3	33	49	PI	P	P	P	P	170	0n MV	N	1M	P	96 on MV	2	56	26	2.15
43	473945	b/o monika	16H	M	4Y	N	39W	LSCS	T	N	N	N	N	N	N	N		6	7	9	2.72	34	49	PI	G	G	G	G	154	62	N	N	P	98	1	18	14	1.14
44	508738	b/o kalpana	3H	F	1Y	N	38W	NVD	T	N	N	N	N	N	N	N		6	8	9	3.5	34	48	PI	G	G	G	G	164	66	N	N	P	86	1	13	12	1.25
45	617508	b/o kavitha	6H	M	4Y	N	39W	NVD	T	N	N	N	N	N	N	N	N	NK	NK	NK	2.68	34	50	PI	P	P	P	G	150	48	N	S	P	85	2	19	10	1.9
46	484135	b/o usha	3H	M	2Y	2	39W6D	LSCS	T	N	N	N	N	N	N	N	N	4	6	6	3	33	49	PI	G	G	G	G	124	32	N	N	P	96	1	17	7	2.4

47	480924	b/o roopa	30M	M	4Y	N	39W6D	NVD	T	N	N	N	N	N	N	N		4	6	8	3	33	49	PI	P	P	P	P	170	0n MV	N	N	P	96 on MV	3	38	8	4.75
48	409393	b/o shilpa	3H	F	1Y	2	39W3D	LSCS	T	N	N	N	N	N	N	N		4	6	6	2.42	32	48	PI	P	P	P	P	170	on MV	N	N	P	96 MV	3	37	7	5.2
49	485644	b/o shamala	4H30M	M	6Y	2	40W 4D	FAD	T	N	N	N	N	N	N	N	N	4	6	7	3.3	33.5	49	PI	M	M	M	M	166	72	N	3M	P	93	2	48	23	2.08
50	525064	b/0 lavanya	5H	F	4Y	N	39W6D	NVD	T	N	N	N	N	N	N	N	N	4	6	6	3	33	49	PI	G	G	G	G	124	32	N	1M	P	96	2	82	65	1.26
51	525549	B/O RUKSANA BEGUM	6H	M	3Y	3	40WW	LSCS	T	N	N	N	N	N	N	N	N	4	6	8	2.5	32	46	PI	G	G	G	G	156	48	N	1M	P	94	2	16	15	1.06
52	324232	b/o swathi	3H	F	2Y	2	39W	NVD	T	N	N	N	N	N	N	N	N	4	6	8	2.5	32	46	PI	G	G	G	G	156	48	N	N	P	94	1	16	15	1.06
53	447330	b/o poolandevi	6H	M	3Y	N	39W6D	LSCS	T	N	N	N	N	N	N	N	N	4	6	8	3	33	49	PI	P	P	P	P	170	48	N	N	P	96	1	16	14	1.14
54	554629	b/o pooja	7H	F	4Y	N	40W 4D	NVD	T	N	N	N	N	N	N	N	N	4	6	7	3.3	33.5	49	PI	G	G	G	G	166	72	N	N	P	93	1	18	16	1.125
55	554613	b/o vani	8H	M	3Y	N	39W6D	LSCS	T	N	N	N	N	N	N	N	N	5	6	6	3	33	49	PI	G	G	G	G	124	32	N	N	P	96	1	82	65	1.26
56	554642	b/o sahana	3H	F	2Y	N	40WW	NVD	T	N	N	N	N	N	N	N	N	5	6	8	2.5	32	46	PI	G	G	G	G	156	48	N	N	P	94	1	16	15	0.64
57	500211	b/o nagarathnamma	3H	F	1Y	N	40W	NVD	T	N	N	N	N	N	N	N	F	4	6	6	3	33	49	PI	P	P	P	P	124	32	N	1M	P	96	3	82	25	3.2
58	571096	b/o fathima shabana	12H	F	2Y	N	40W	NVD	T	N	N	N	N	N	N	N	N	NK	NK	NK	2.8	34	49	PI	P	P	P	P	170	40	N	1M	P	70	3	32	9	3.5
59	516980	b/o hamsa	30M	F	4Y	N	39W	LSCS	T	N	N	N	N	Y	N	N	N	3	5	NK	3.86	33	46	PI	G	P	P	P	160	58	N	1M	P	68	3	30	10	3
60	566533	b/o amaravathi	12H	F	4Y	N	40W	NVD	T	N	N	N	N	Y	N	N	N	NK	NK	NK	2.75	35	46	PI	P	P	P	P	170	G	N	1M	P	64	3	49.5	15.4	3.2
61	566533	b/o amaravathi	12H	F	4Y	N	40W	NVD	T	N	N	N	N	Y	N	N	N	NK	NK	NK	2.75	35	46	PI	P	P	P	P	170	G	N	1M	P	64	2	1.6	4	0.4
62	553885	b/o jyothi	16H	F	4Y	N	39W	NVD	T	N	N	N	N	Y	N	N	N	NK	NK	NK	1.92	33	38	PI	G	G	G	G			N	N	P		1	170	100	1.7
63	573474	b/o shabana	24H	M	5Y	N	40W	NVD	T	N	N	N	N	Y	N	N	N	3	5	NK	3.2	33	49	PA	P	P	P	P	170	72	N	1M	P		3	22	4	5.5
64	586058	b/o jyothi	12H	M	3Y	N	40W	NVD	T	N	N	N	N	N	N	N	N	NK	NK	NK	3	35	50	PI	G	G	G	G	140	44	N	1M	P	93	2	20	10	2
65	577388	b/o yasmin	12H	M	5Y	N	37W	LSCS	T	N	N	N	N	N	N	N	IDM,HM	NK	NK	NK	3.5	33	48	PI	P	P	P	P	160	48	N	1M	P	90	3	20	6	3.33
66	571528	b/o anithamma	4H	M	4Y	N	40W	NVD	T	N	N	N	N	N	N	N	N	NK	NK	NK	3	32	48	PI	P	P	P	P	170	66	N	1M	P	92	3	28	6	4.6
67	553036	b/o manjula	16H	F	5Y	N	40W	LSCS	T	N	N	N	N	Y	N	N	N	NK	NK	NK	3	33	50	PI	P	P	P	P	10	66	N	1M	P	92	2	14.9	8	1.86
68	565019	b/o salma jan	3H	M	5Y	N	38W	LSCS	T	N	N	N	N	N	N	N	N	NK	NK	NK	2.9	33	48	PI	G	P	P	P	162	64	N	1M	P	60	3	64	14	4.5
69	585499	b/o roja	24H	M	4Y	N	40W	NVD	T	N	N	N	N	Y	N	N	N	NK	NK	NK	3	33	48	PI	P	P	P	P	170	G	N	1M	F	65	3	52.8	12.1	4.36
70	559109	b/o sumithra	23H	F	3Y	N	40W	NVD	T	N	N	N	N	Y	N	N	N	NK	NK	NK	2.9	33	49	PI	G	G	G	G	140	44	N	1M	P	94	2	58	220	0.26
71	584694	b/o gowthami h v	2H	F	3Y	N	40W	NVD	T	N	N	N	N	Y	N	N	N	NK	NK	NK	1.75	32	44	PI	G	G	G	G	148	44	N	N	P	97	1	170	82	2.07
72	563716	b/o neelamma	12H	F	3Y	N	37W	NVD	T	N	N	N	N	N	N	N	N	NK	NK	NK	1.86	32	48	C	P	P	P	P	102	23	N	1M	P	64	3	63	20	3.15
73	564039	b/o vimala	4H	F	3Y	N	40W	FAD	T	N	Y	N	N	N	N	N	N	NK	NK	NK	3	33	49	PI	P	P	P	P	162	63	N	1M	P	95	3	28	10	2.8
74	571528	b/o anithamma	4H	M	4Y	N	40W	NVD	T	N	N	N	N	N	N	N	N	NK	NK	NK	3	32	48	PI	P	P	P	P	170	66	N	1M	P	92	2	24	8	3
75	572312	b/o sindhu	4H	M	3Y	N	40W	NVD	T	N	N	N	N	Y	PROM	N	N	3	6	NK	3	32.5	48	PI	P	P	P	P	160	68	N	1M	P	94	3	30	12	2.5
76	572814	b/o devi	20H	F	2Y	N	38W	LSCS	T	N	N	N	N	N	N	N	H/O F ,IDM	NK	NK	NK	3.46	32	48	PI	G	G	G	G	170	68	N	N	P	94	1	28	10	2.8
77	494445	b/o arshiya taj	23H	F	3Y	N	40W	NVD	T	N	N	N	N	N	N	N	N	NK	NK	NK	3.3	33	50	PI	G	G	G	G	140	46	N	N	P	97	1	4	14	0.28
78	500235	b/o vasantha	24H	M	3Y	N	40W	NVD	T	N	N	N	N	N	N	N	N	NK	NK	NK	3	33	50	PI	G	G	G	G	140	46	N	N	P	97	1	128	78	1.64
79	497728	b/o nagma khathun	24H	M	3Y	N	40W	NVD	T	N	N	N	N	N	N	N	N	NK	NK	NK	3	33	50	PI	G	G	G	G	140	46	N	N	P	97	1	126	76	1.65
80	508376	b/o chandu	23H	F	2Y	N	40W	NVD	T	N	N	N	N	N	N	N	N	NK	NK	NK	3.23	35	52	PI	P	P	P	P	122	G	N	1M	P	65	3	12.6	3.8	3.31
81	519957	b/o annaPneswari	24H	M	1Y	N	38W	NVD	T	N	N	N	N	N	N	N	N	NK	NK	NK	3.2	34	51	PI	P	P	P	P	144	44	N	1M	P	96	2	52	21	2.47
82	594851	b/o shobha	34H	F	2Y	N	40W	NVD	T	N	N	N	N	Y	N	N	N	NK	NK	NK	2.64	33	48	PI	G	G	G	G	150	55	N	N	P	98	1	66	30	2.2
83	517503	b/o nirmala	3D	M	3Y	N	40W	NVD	T	N	N	N	N	N	N	N	N	NK	NK	NK	2.96	33	49	PI	P	P	P	P	140	48	N	1M	P	94	3	24.3	8.5	2.8
84	523801	b/o ashwitha	30M	M	2Y	N	40W	LSCS	T	N	N	N	N	Y	N	N	N	NK	NK	NK	3.244	33	49	PI	G	G	G	G	160	58	N	N	P	96	1	79	23.5	3.36
85	523388	b/o uma	24H	F	3Y	N	40W	NVD	T	N	N	N	N	N	N	N	N	NK	NK	NK	3	33	48	PI	G	G	G	G	160	68	N	1M	P	90	2	28	11.5	2.43
86	441323	b/o radhika	23H	F	3Y	N	40W	LSCS	T	N	N	N	N	Y	N	N	N	NK	NK	NK	3.06	33	48	PI	P	P	P	P	154	68	N	1M	P	85	3	124	36	3.44
87	594755	b/o lakshmi	30M	M	2Y	N	40W	LSCS	T	N	N	N	N	Y	N	N	N	4	8	NK	3.82	35	52	PA	G	G	G	G	150	70	N	1M	P	96	2	76	32	2.3
88	562883	b/o radhamma	24H	M	3Y	N	40W	NVD	T	N	N	N	N	N	N	N	N	NK	NK	NK	2.44	33	48	PI	P	P	P	P	162	68	N	1M	P	88	3	13.6	2.5	5.44
89	586919	b/o sujatha	6D	M	1Y	N	38W	LSCS	T	N	N	N	N	Y	N	N	N	NK	NK	NK	2.75	32	48	PI	G	G	G	G	170	70	N	1M	P	96	2	13	6.5	2
90	579468	b/o hemavathi	30M	M	1Y	N	38W	LSCS	T	N	N	N	N	N	N	N	RU	1	3	4	2.5	33	48	C	P	P	P	P	50	0	N	1M	P	80	3	42	11	3.8
91	575323	b/o sujatha	4H	M	2Y	N	40W	NVD	T	N	N	N	N	N	N	N	N	3	5	NK	3	33	48	PI	P	P	P	P	170	66	N	1M	P	96	3	28	8	3.5
92	589677	b/o almaz	24H	M	3Y	N	37W	LSCS	TERM	N	N	N	N	N	N	N	N	NK	NK	NK	2.9	33	48	PI	P	P	P	P	118	70	N	1M	P	96	2	58	12	4.83
93	584678	B/O NALINI	1H	M	3Y	N	40W	FAD	T	N	N	N	N	Y	N	N	N	NK	NK	NK	1.86	34	48	C	G	G	G	G	160	68	N	1M	P	97	2	37.4	48.4	0.77
94	551146	b/o shaheena	12H	M	2Y	N	40W	NVD	T	N	N	N	N	N	N	N	N	NK	NK	NK	3	35	49	PI	G	G	G	G	148	56	N	1M	P	95	2	138	57	2.42
95	546310	b/o umerabegum	12H	M	2Y	N	38W	NVD	T	N	N	N	N	N	N	N	N	NK	NK	NK	2.6	33	48	PI	P	P	P	P	150	60	N	1M	P	90	3	45	9.9	4.5
96	582729	b/o kavya	2H	M	2Y	N	40W	LSCS	T	N	N	N	N	N	PROM	N	N	NK	NK	NK	2.5	33	48	C	G	G	G	G	170	70	N	N	P	99	1	83	21	3.95
97	590657	B/O SHABIN TAJ	16H	M	4Y	N	40W	NVD	T	N	N	N	N	N	PROM	N	N	NK	NK	NK	2.5	33.5	50	PI	G	G	G	G	158	68	N	N	P	96	1	40	12	3.3

98	517994	b/o manjula	24H	M	3Y	N	39W	NVD	T	N	N	N	N	N	N	N	N	NK	NK	NK	2.8	33	50	PI	G	G	G	G	160	66	N	1M	P	92	2	37	17	2.17
99	517994	b/o manjula	49H	M	3Y	N	39W	NVD	T	N	N	N	N	N	N	N	N	NK	NK	NK	2.8	33	50	PI	P	P	P	P	160	66	N	1M	P	85	3	75.5	13.5	5.59
100	619554	b/o nehaanjum	6H	M	3Y	N	39W	NVD	T	N	N	N	N	N	N	N	N	NK	NK	NK	3	33	50	PI	G	G	G	G	158	66	N	1M	P	94	2	26	11	2.36
101	612388	b/o thaslimataj	2H	M	3Y	N	41W	NVD	PD	N	N	N	N	N	N	N	N	NK	NK	NK	3.5	33	50	PI	G	G	G	G	160	68	N	1M	P	92	2	39.6	25.3	2.375
102	615442	b/o sangeetha	24H	M	1Y	N	39W	NVD	T	N	N	N	N	N	N	N	N	NK	NK	NK	3	33	50	PI	P	P	P	P	170	54	N	S	P	85	3	26.5	6.8	3.89
103	615370	b/o lavanya	24H	M	5Y	N	40W	NVD	T	N	N	N	N	N	N	N	N	NK	NK	NK	3.02	34	50	PI	P	P	P	P	160	70	N	S	P	88	3	32	12	2.6
104	599690	b/o lalitha k	30M	F	2Y	N	39W	LSCS	T	N	N	N	N	N	N	N	N	NK	NK	NK	2.64	33	50	PI	G	G	G	G	154	64	N	N	P	96	1	99	45	2.19
105	597018	b/o aruna	10H	M	5Y	N	39W	NVD	T	N	N	N	N	N	N	N	N	NK	NK	NK	2.8	33	49	PI	G	G	G	G	144	44	N	1M	P	98	2	20	10	2
106	596918	b/o shashikala	6H	M	2Y	N	40W	NVD	T	N	N	N	N	Y	N	N	N	NK	NK	NK	2.74	33	50	PI	G	G	G	G	160	47	N	N	P	98	1	62	38	1.63
107	616762	b/o radhika	24H	M	3Y	N	38W	NVD	T	N	N	N	N	N	N	N	N	NK	NK	NK	2.68	33	48	PI	G	G	G	G	140	48	N	1M	P	96	2	14	5	2.8
108	616430	b/o anushree	48H	F	3Y	N	39W	LSCS	T	N	N	N	N	N	N	N	N	NK	NK	NK	2.25	32	49	PI	G	G	G	G	138	49	N	1M	P	95	2	28	19	1.47