PREVALENCE OF HYPOGLYCEMIA AMONG CHILDREN ADMITTED AT KENYATTA NATIONAL HOSPITAL.

A research proposal submitted in partial fulfillment for the degree of Master of Medicine in Paediatrics and Child Health at the University on Nairobi.

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DECLARATION

This dissertation proposal is my original work and has not been presented for the award of a degree in any other university.

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M.B.Ch.B (University of Nairobi)
Signed…………………………………   Date   ……………………………….

This dissertation proposal has been presented with full approval of supervisors.

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- All the babies and their caregivers who participated in the study process for their patience and co-operation.
- The staff at the pediatric filter clinic: the nurses, doctors on call during the study period and support staff for their help and input throughout the study process.
DEDICATION

I dedicate this book to my entire family for their support throughout the study period.
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ABBREVIATIONS

EGP  - Endogenous Glucose Production

PEM  - Protein Energy Malnutrition

PFC  - Pediatric Filter Clinic

KNH  - Kenyatta National Hospital

WHO  - World Health Organization

RBS  - Random Blood Sugar

ETAT - Emergency Triage and Treatment
DEFINITIONS

Level of consciousness: 
A- Alert
V- Child responding to verbal stimulation.
P- Child responding to pain stimulation.
U- Unresponsive child.

Diarrhoea – The passage of 3 or more loose or liquid stools per day, or more frequently than is normal for the child.

Convulsion – Uncontrollable muscular contractions that make the body shake rapidly.

Z-score – Weight in kg/ length in meters.

Traditional medicine – Alternative or complementary medicines used in maintenance of health or treatment of illness based on the theories and beliefs indigenous to different cultures.

No dehydration – Diarrhoea/ Gastroenteritis with fewer than 2 signs of dehydration namely: sunken eyes, return of skin pinch 1-2 seconds, restlessness/ irritability.

Some dehydration – Ability to drink adequately but with 2 or more of: sunken eyes, return of skin pinch 1-2 seconds, restlessness/ irritability.

Severe dehydration – Inability to drink or AVPU <A plus: sunken eyes and return of skin pinch ≥ 2 seconds.

Shock – Cold hands plus weak/ absent pulse and either: capillary refill> 3 seconds or AVPU< A.

Sick – physically or mentally ill, not well or healthy.
ABSTRACT

Introduction
Hypoglycemia is a common metabolic derangement in childhood that if not treated promptly, has a high risk of causing brain damage and is an independent risk factor for death.

Hypoglycemia also complicates common childhood illnesses. Children are most susceptible because of their developing brain and its dependence on glucose as its primary fuel.

Study design
This is a descriptive hospital-based cross-sectional study aimed at determining the prevalence of hypoglycemia in sick children admitted at Kenyatta National Hospital.

Study Objectives
The primary objective was to determine the prevalence of hypoglycemia in sick children requiring admission aged between 2 months and 5 years. The secondary objectives were to determine the risk factors associated with hypoglycemia and the outcome at 24 hours of admission.

Methodology
Consecutive enrolment of patients who satisfied the study criteria had blood samples taken by heel prick following aseptic measures and analyzed using the Hemocue® glucose analyzer. A questionnaire was issued to the parent or guardian after performing resuscitation measures on the child where necessary and a physical examination done thereafter. The outcome of the child at 24 hours after admission was then documented.

Results
Four out of the 111 children enrolled into the study were found to have hypoglycemia with a prevalence of 3.6%. However, five children (4.5%) were found to be hyperglycemic.

Hypoglycemia was found to complicate various childhood illnesses. None of the factors assessed such as fasting more than 12 hours, childhood diseases such as Malaria, Pneumonia, Meningitis, Gastroenteritis and Malnutrition was found to be statistically significantly associated with hypoglycemia.

Similarly, the presence of hypoglycemia was not significantly associated with the outcome at 24 hours of admission.
1.0 BACKGROUND AND LITERATURE REVIEW

1.1 INTRODUCTION

Hypoglycemia in childhood is one of the most common metabolic abnormalities that needs to be treated promptly or prevented because it is a frequent cause of neurological damage or death (1, 2).

WHO defines hypoglycemia as blood glucose levels <1.1, <2.5, and <3.0 mmol/ltr depending on different clinical categories as defined below (3, 4).

<table>
<thead>
<tr>
<th>Clinical category</th>
<th>BGC (Blood glucose conc.)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Newborn/Infant/Child with ‘signs of illness’</td>
<td>&lt;2.5mmol/ltr or 45mg/dl</td>
</tr>
<tr>
<td>Healthy term/preterm newborn- ‘feeding well’</td>
<td>&lt;1.1mmol/ltr or 19.8mg/dl</td>
</tr>
<tr>
<td>Infant/Child with severe malnutrition</td>
<td>&lt;3.0mmol/ltr or 54mg/dl</td>
</tr>
</tbody>
</table>

The child’s developing brain is more susceptible to hypoglycemia than the adult brain (1, 2, 5). Although it can use lactate, ketone bodies and amino acids, its primary fuel is glucose, thus making it highly vulnerable to low plasma glucose levels during fasting (6). Recurrent hypoglycemia may result in permanent neurological damage (2, 5, 7). The major long-term sequela of severe prolonged hypoglycemia is neurological damage resulting in mental retardation, transient cognitive impairment, neurological deficit and recurrent seizures.

Young age, fasting, and severe infectious diseases are considered important risk factors for the development of hypoglycemia (8).

In the fasting state, the plasma glucose level is maintained within the lower normal range by a delicate balance between endogenous glucose production (EGP) through gluconeogenesis and glycogenolysis on one hand and glucose utilization on the other hand.

Children have a limited tolerance to fasting because they have less glycogen stores. They are able to maintain a normal plasma glucose level during a fasting period of only 12 hours (9,10).
Healthy adults are able to maintain normal plasma glucose levels for up to 86 hours of fasting (11). Many infectious diseases are characterized by starvation due to disease-induced anorexia as well as cultural customs and traditional restriction of feeding in disease (12).

Several studies suggest that fasting is a major factor in the occurrence of hypoglycemia in children because of an association between the occurrence of hypoglycemia and the time since the last meal (13-17). Children presenting after 12 hours of their last meal are more likely to be hypoglycemic compared to those presenting within 12 hours of their last meal.

Studies have been done to determine the prevalence of hypoglycemia in paediatric emergency admissions. Elusiyan et al (17) found a prevalence of 6.4% in a Nigerian paediatric emergency ward while Solomon et al (15) obtained a prevalence of 7.1% among paediatric admissions in an urban hospital in Mozambique. Osier et al (13) found a prevalence of 7.3% among pediatric admissions in a rural district Kenyan hospital, whereas Pershad et al (18) obtained a prevalence of 6.54% of 100000 visits in an urban emergency department in the U.S.A.

The above studies show that hypoglycemia is a common childhood problem with an almost similar prevalence worldwide.

In all these studies, hypoglycemia was found to be associated with an increase in mortality and was also shown to complicate many childhood illnesses.

Elusiyan et al (17) found that of the total number of deaths, 28.6% had hypoglycemia against 4.2% of the normoglycemic children who died. In the study by Osier et al (13), mortality in hypoglycemic children was found to be 20.2% compared to 3.8% found in normoglycemic children. Solomon et al (15) found that of the children who died, 16.3% had hypoglycemia compared to 3.2% of the normoglycemic children.

1.2 HYPOGLYCEMIA AND MALARIA

Hypoglycemia has been found to complicate many childhood illnesses such as malaria, pneumonia, protein energy malnutrition (PEM) as well as diarrhoeal diseases (13, 15-17, 19-21).

In a Nigerian study (17), 44% of the hypoglycemic cases were suffering from severe malaria, compared to 5.2% reported by Solomon et al (15) in Mozambican children and 16% in Kenyan
children as reported by English et al (16). A frequency of 11.6% of hypoglycemia in malaria cases was found in Togo children (22) and 11.4% in Ugandan children (23) with malaria.

The highest prevalence rates of hypoglycemia among children with malaria are the 31% reported in Gambian children (24) and 20% in Malawian children (25).

In African children with malaria, impairment in hepatic gluconeogenesis in the presence of adequate precursors has been considered to be the most likely mechanism in the causation of hypoglycemia.

English et al (16) found that hypoglycemia tends to occur in children who are more acidic and who have greater evidence of renal impairment. Acidosis is known to inhibit hepatic uptake of lactate at high concentrations (>2mmol/ltr) and to impair gluconeogenesis. The normal kidneys on the other hand, partly compensate for this reduction in hepatic metabolism of lactate under conditions of acidosis with an increase in fractional lactate uptake. Such a compensatory mechanism may be lost if renal function is impaired.

Increased glucose consumption by malaria parasites is considered to be a contributing factor. Parasitized erythrocytes consume up to 30-75 times the quantity of glucose consumed by non-infected cells. Another well known contributing factor is the stimulation of insulin release in vivo by quinine and quinidine administration in malaria treatment with resultant hyperinsulinemic hypoglycemia (26).

Cerebral malaria is also a leading contributor to hypoglycemia. The sequestration of parasitized red cells in the venules and capillaries of deep tissues has been implicated in the pathogenesis of both cerebral malaria and hypoglycemia (27). Hypoglycemia is an important feature in children with malaria because it predicts an increased mortality (28, 29). The mortality rate increases 4-6 fold in children with malaria complicated with hypoglycemia (13, 28, 29). 20-30% of the children admitted with malaria who had hypoglycemia did not survive compared with a mortality rate of 3.8% in normoglycemic children (13). Thus, hypoglycemia is considered to be one of the major predictors of adverse outcome in children with severe malaria.

Hypoglycemia does not only occur in children with malaria but is also seen in children with other infectious diseases such as pneumonia and diarrhoea, both in tropical (13-15,17,19-21) and
in Western countries (30,31). In a Kenyan rural district hospital, Osier et al (13) found hypoglycemia in 8.2% of all children seen in the hospital, with the highest prevalence occurring in neonates and children between 2 and 4 years.

The proportion of children with hypoglycemia varied significantly with the primary diagnosis, with the highest proportion being observed in children with meningitis at 36%. The proportion in children with malaria was 8.4%. Multiple logistic regression analysis identified prostration, deep breathing, severe malnutrition, fasting of more than 12 hours and a positive malaria slide as independent indicators of hypoglycemia in the same study.

1.3 HYPOGLYCEMIA AND PNEUMONIA

Elusiyan et al (17) found septicemia to be the second most frequent diagnosis associated with hypoglycemia at 24%, after malaria at 44%. Most of the diagnoses of septicemia in the same study were based on clinical assessment with only a few bacteriological confirmations. Solomon et al (15) found pneumonia as the leading diagnosis associated with hypoglycemia at 21% while a Nigerian study found pneumonia associated with hypoglycemia to be at 8.6% (17).

1.4 HYPOGLYCEMIA AND GASTROENTERITIS

Diarrhoeal diseases represent a major health problem in developing countries and also a high risk to travellers visiting these countries. It is estimated that the global death toll from diarrhoeal disease is about 2 million deaths per year (1.7-2.5 million deaths), ranking third among all causes of infectious disease deaths worldwide.

Huq et al (21) performed a prospective study in under-five children who presented with diarrhoeal illnesses and found that 11% of those children had hypoglycemia. Among hypoglycemic children, 11% had bacteremia while among the non-hypoglycemic children, only 5% had bacteremia. In this study, the case fatality rate was proportionately higher in children who had both bacteremia and hypoglycemia (43%) compared to those who had bacteremia, but not hypoglycemia (30%).

Decreased stores of glycogen, impaired gluconeogenesis, increased peripheral utilization of glucose and intestinal malabsorption have all been associated with hypoglycemia. In children, hypoglycemia is associated with deaths from infectious diarrhoea regardless of their nutritional
status (32). Bacteremic children with clinical sepsis were found to be 4 times more likely to develop hypoglycemia. This study concluded that a rapid bedside blood glucose test should be considered as an inexpensive alternative in the management decisions of diagnosing bacteremia and initiating empiric antibiotic treatment in children with diarrhoeal disease. In the Kenyan study (13), 5.5% of children with hypoglycemia had gastroenteritis and in another study by Nyikuri et al (33) in Kenyatta National Hospital, 4.2% of children with acute diarrhoea had hypoglycemia.

1.5 HYPOGLYCEMIA AND MALNUTRITION

Severe malnutrition is a known cause of hypoglycemia. WHO defines hypoglycemia in severe malnutrition as a blood glucose concentration below 3mmol/ltr (54mg/dl). Nzioki et al (34) performed an audit of care for children aged 6-59 months admitted with severe malnutrition at Kenyatta National Hospital, and found that diagnosis, treatment and prevention of hypoglycemia were inadequately done both at the pediatric filter clinic (PFC) and the wards.

Malnourished children have diminished glycogen stores and may not be able to mobilize hepatic gluconeogenic substrates.

Indeed Step 1 of the WHO guidelines on care of severely malnourished children is the treatment of hypoglycemia (35). Nzioki et al (34) found that out of the 100 children in the study, only 29.9% had a rapid blood glucose test done in either PFC or the wards despite the tools needed being largely available.

There was also a long delay in initiating feeds in the wards with a median waiting time of 14.7 hours. 34% of patients overall received their initial feed after 19 hours of admission to the ward.

In the Nigerian study (17), one (50%) of the two cases of Kwashiorkor had hypoglycemia but the two patients that died from PEM in this study both had hypoglycemia. In the Kenyan study (13), mortality was high at 31.8% in hypoglycemic children who had signs of severe illness (prostration or deep breathing) on admission or severe malnutrition while in normoglycemic children the mortality was found to be 9%. Hypoglycemia is, thus, associated with childhood illnesses and increased mortality. Currently, the only local sources of data on prevalence of hypoglycemia are that of a study by Nyikuri et al on the prevalence of hypoglycemia in childhood diarrhoeal cases. This study will determine the prevalence of hypoglycemia in an
urban referral centre as well as describe the risk factors associated with hypoglycemia. It will also determine the outcome of the patients at 24 hours of admission.
1.6 STUDY JUSTIFICATION AND UTILITY

Hypoglycemia is a common and serious condition associated with brain damage and an increase in mortality, yet it is amenable to inexpensive and widely available treatment. It is a frequent but poorly explored feature of severe infectious diseases and malnutrition in children.

Children have a limited tolerance to low blood sugar because they have less glycogen stores and are therefore unable to maintain normal plasma glucose after a prolonged fasting period.

A study by Nzioki et al (13) at KNH showed that only 29.9% of children seen either in PFC or in the wards had a rapid blood glucose test done despite the tools needed being largely available. Out of these, 13.3% were found to have hypoglycemia. The locally available data on the prevalence of hypoglycemia is restricted to childhood diarrhoea cases in a study done by Nyikuri et al in 1994 (33).

It is hoped that this study will provide data on the prevalence of hypoglycemia among sick children aged 2 months to 5 years. Such data will be useful for developing a policy on evaluation of all sick children for hypoglycemia and its prompt management with the aim of reducing morbidity and mortality.
2.0 STUDY OBJECTIVES

2.1 Primary objective:
To determine the prevalence of hypoglycemia in sick children requiring admission into Kenyatta National Hospital.

2.2 Secondary objectives:

1. To determine some of the factors that may be associated with hypoglycemia. (Potential risk factors include fasting more than 12 hours, drugs such as Quinine, childhood illnesses such as Malaria, Pneumonia, Meningitis, Gastroenteritis and Malnutrition).

2. To determine the effect of hypoglycemia on the outcome at 24 hours of admission (Dead or Alive).
3.0 RESEARCH METHODOLOGY

3.1 STUDY DESIGN
This was a hospital based descriptive Cross-Sectional study.

3.2 STUDY AREA
Kenyatta National Hospital (KNH) serves as the national referral hospital. It is an urban tertiary teaching hospital that serves a population of mainly medium to low socio-economic status. The study was conducted at the Paediatric Filter Clinic (PFC) where all children requiring admissions are seen before transfer to the wards. Subsequent follow up was done in the wards to determine the outcome at 24 hours of admission.

3.3 STUDY PERIOD
The study was conducted over a period of 3 months (September- November 2012).

3.4 STUDY POPULATION
All sick children aged 2-60 months requiring admission whose parent/guardian gave informed consent were recruited into the study.

3.5 INCLUSION CRITERIA
Sick children aged 2-60 months requiring admission.

Children whose parent/guardian gave informed consent to participate in the study.

3.6 EXCLUSION CRITERIA
Children known to have Type 1 Diabetes Mellitus.

Children referred from other hospitals having received initial treatment for hypoglycemia elsewhere within 12 hours of arrival.

Children whose parent/guardian did not give consent.
3.7 SAMPLE SIZE CALCULATION

The sample size was calculated using the Fisher’s formula for Prevalence studies.

\[ n = \frac{Z^2 PQ}{D^2} \]

Where:

- \( n \) = desired sample size.
- \( Z \) = standard deviation which corresponds to the 95% confidence level (1.96).
- \( P \) = the estimated prevalence as reported by Osier et al in Kenya was 7.3 % (13)
- \( Q \) = \( 1 - P \) =1-0.05 =0.95
- \( D \) = the degree of accuracy desired, set at 0.05.

Hence:

\[ n = \frac{1.96^2 \times 0.073 \times 0.95}{0.05^2} \]

\[ = 106 \]
3.8 SAMPLING TECHNIQUE

The children who satisfied the inclusion criteria were consecutively recruited into the study after obtaining informed consent.

3.9 DATA COLLECTION

- Sick children were triaged and those who were decided for admission by the primary admitting resident paediatrician were recruited into the study if they satisfied the inclusion criteria and consent given by the parent on a predesigned form.
- A structured questionnaire was then issued to the parent to get information such as the bio data, factors associated with hypoglycaemia such as the duration of illness and the patient’s last meal. Additional information included the symptoms of the illness and the medication given prior to arrival at the hospital.
- A clinical examination of the child was done by the principal investigator or the research assistant, with the help of a structured clinical bio data form. This included temperature, measurements of weight using a weighing scale, and length or height using a stadiometer so as to determine the Z score (weight for height). A general assessment of the child was also done to assess the factors associated with hypoglycaemia. The primary clinical diagnosis made by the attending doctor was documented in the clinical bio data form.

3.10 COLLECTION OF BLOOD SAMPLES

- Blood samples were taken by heel prick under strict sterile measures. The foot was warmed before sample collection to allow sufficient vasodilatation hence free flow of the required amount of blood without having to compress the foot.
- The heel of the foot was then sterilized with 70% alcohol swabs and pricked using new lancets for each prick. Five micro litres of blood was collected into microcuvettes which were then fed into the Hemo-Cue® glucose analyzer for a reading to be taken. A dry cotton swab was then placed on the affected area of the foot and gentle pressure applied to the area until blood stopped oozing.
- Plasma glucose levels were determined using the Hemo-cue beta glucose analyzer. The Hemo-cue beta glucose analyzer uses a dual wave length photometer to measure glucose in hemolysed whole blood after a modified glucose dehydrogenase reaction using dried
reagents contained in disposable micro-cuvettes. It is portable and very useful as a point of care device (36). The instrument can use whole blood or plasma without additional processing and has been shown to be reliable, accurate and sensitive in the clinical setting and for research purposes (37). It is convenient and technically easy to use requiring very little maintenance (38). The instrument uses only 5 micro litres of blood drawn up by capillary action into a cuvette. This makes it safe to use as sampling of larger amounts of blood (1 or 2ml) for conventional laboratory analysis may contribute to already existing anaemia. Its accuracy and precision is comparable to that of conventional laboratory instrumentation (38). The analyzers are factory calibrated and require minimal maintenance. No calibration is required between cuvette batches (39).
3.11 DATA ANALYSIS

Data was analyzed using SPSS version 17.0 software. It was coded, cleaned and summarized using tables for the different variables. Data was presented using graphs, pie charts and tables. Descriptive statistics of individual variables including means, percentages, standard deviations and standard error was analyzed. Analytical statistics included relative prevalence, Chi-square and student t-test to test for the strength of association between the variables.

3.12 DISSEMINATION OF RESULTS

The findings of the study were distributed to University of Nairobi – Paediatrics department, Kenyatta National Hospital and copies made available in the University of Nairobi library. The results were made available to policy makers and health workers within the hospital and university to facilitate improvement of health services offered to children admitted at KNH.
3.13 ETHICAL CONSIDERATIONS

Approval to carry out the study was sought from Kenyatta National Hospital Medical Review Board and Ethics Committee.

Informed written consent to participate in the study was obtained from the parent or primary caregiver accompanying the child after explanation of the study and voluntary nature of participation. Caregivers were informed that refusal to participate in the study would not affect the treatment of their child.

Extreme care was taken while undertaking the procedure of sample collection, which was done under sterile conditions.

Any information pertinent to the management of the child discovered during the interview or blood glucose determination was communicated to the primary attending doctor.

Any child found to be hypoglycaemic was given a bolus of 5mls/kg of 10% dextrose intravenously followed by feeds if able to take orally or by dextrose saline solution as a maintenance fluid. Hyperglycaemic children had their blood sugars monitored in the wards. No intervention was undertaken at the filter clinic.

Confidentiality was maintained.

3.14 CONTROL OF ERRORS AND BIAS

The questionnaire was pretested on a sample population to ensure validity before commencement of the study.

The research assistant was trained and provided with standard definitions of terminologies used in the questionnaire to ensure uniform interpretation.

Data collected was entered daily into a pre-programmed computer. The data entered was crosschecked to ensure validity and completeness.
4.0 RESULTS

One hundred and eleven children admitted to KNH were recruited into the study.

4.1 Characteristics of the study population

The study subjects recruited were aged between 2 and 60 months, 56 (50.4%) were males while 55 (49.5%) were females. The median age was 12 months with an interquartile range of (7.0-24.0) months. Table 1 below shows the nutritional status and temperature recordings of the children recruited into the study as part of the assessment of factors associated with hypoglycemia. 21 (18.9%) children were found to have severe malnutrition with a Z-score of -3SD and below. 49(44.1%) children had Z-scores of -2SD and below while 28 (25.2%) children had a Z-score of between-2SD and -3SD. 2 (1.8%) children, however, had missing data. 35(31.5%) children were febrile with temperatures above 38.6°C while 75(67.5%) children had temperatures below 37.5°C. 1 child had missing data. The clinical examination findings of all the 111 children as they were examined are also represented in the table below. 22(19.8%) children had an AVPU less than P, while 25(22.5%) children and 8(7.2%) children had severe dehydration and shock respectively.

The characteristics of the study population are as shown in Table 1 below.
Table 1: Characteristics of the study population

<table>
<thead>
<tr>
<th>Variable</th>
<th>Frequency (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Median age (months)</td>
<td>12.0</td>
</tr>
<tr>
<td>Interquartile range (IQR) in months</td>
<td>(7.0 -24.0)</td>
</tr>
<tr>
<td>Range (months)</td>
<td>2.0 - 60.0</td>
</tr>
<tr>
<td><strong>Sex</strong></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>56 (50.4)</td>
</tr>
<tr>
<td>Female</td>
<td>55 (49.5)</td>
</tr>
<tr>
<td><strong>Variable</strong></td>
<td></td>
</tr>
<tr>
<td>Mean weight (SD)</td>
<td>8.9 (3.8)</td>
</tr>
<tr>
<td>Mean height (SD)</td>
<td>77.1(15.0)</td>
</tr>
<tr>
<td><strong>WHZ score</strong></td>
<td></td>
</tr>
<tr>
<td>&lt;-1SD</td>
<td>35 (31.5)</td>
</tr>
<tr>
<td>-1SD to -2SD</td>
<td>25 (22.5)</td>
</tr>
<tr>
<td>-2SD to -3SD</td>
<td>28 (25.2)</td>
</tr>
<tr>
<td>-3SD to -4SD</td>
<td>14 (12.6)</td>
</tr>
<tr>
<td>&gt;-4SD</td>
<td>7 (6.3)</td>
</tr>
<tr>
<td>Missing</td>
<td>2 (1.8)</td>
</tr>
<tr>
<td><strong>Temperature</strong></td>
<td></td>
</tr>
<tr>
<td>35.0-36.5</td>
<td>20 (18.0)</td>
</tr>
<tr>
<td>36.6-37.5</td>
<td>55 (49.5)</td>
</tr>
<tr>
<td>38.6-40.0</td>
<td>32 (28.8)</td>
</tr>
<tr>
<td>&gt;040</td>
<td>3 (2.7)</td>
</tr>
<tr>
<td>Missing</td>
<td>1 (0.9)</td>
</tr>
<tr>
<td><strong>Duration of illness before admission (days)</strong></td>
<td></td>
</tr>
<tr>
<td>&lt;01</td>
<td>13 (11.7)</td>
</tr>
<tr>
<td>01-7</td>
<td>56 (50.5)</td>
</tr>
<tr>
<td>07-30</td>
<td>30 (27.0)</td>
</tr>
<tr>
<td>&gt;30</td>
<td>12 (10.8)</td>
</tr>
<tr>
<td><strong>Time from the last meal at admission (hours)</strong></td>
<td></td>
</tr>
<tr>
<td>&lt;012</td>
<td>85 (76.6)</td>
</tr>
<tr>
<td>&gt;012</td>
<td>26 (23.4)</td>
</tr>
<tr>
<td><strong>Presence of:</strong></td>
<td></td>
</tr>
<tr>
<td>Fever</td>
<td>91 (82.0)</td>
</tr>
<tr>
<td>Cough</td>
<td>81 (73.0)</td>
</tr>
<tr>
<td>Difficulty in breathing</td>
<td>59 (53.2)</td>
</tr>
<tr>
<td>Vomiting</td>
<td>37 (33.3)</td>
</tr>
<tr>
<td>Convulsions</td>
<td>58 (52.3)</td>
</tr>
<tr>
<td>Diarrhoea</td>
<td>45 (40.5)</td>
</tr>
<tr>
<td>Level of consciousness</td>
<td></td>
</tr>
<tr>
<td>-------------------------</td>
<td>---</td>
</tr>
<tr>
<td>A</td>
<td>83 (74.8)</td>
</tr>
<tr>
<td>V</td>
<td>6 (5.4)</td>
</tr>
<tr>
<td>P</td>
<td>19 (17.1)</td>
</tr>
<tr>
<td>U</td>
<td>3 (2.7)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Dehydration</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>No dehydration</td>
<td>64 (57.7)</td>
</tr>
<tr>
<td>Some dehydration</td>
<td>14 (12.6)</td>
</tr>
<tr>
<td>Severe dehydration</td>
<td>25 (22.5)</td>
</tr>
<tr>
<td>Shock</td>
<td>8 (7.2)</td>
</tr>
</tbody>
</table>

<p>| | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Able to drink/ breastfeed</td>
<td>80 (72.1)</td>
</tr>
<tr>
<td>Deep breathing</td>
<td>41 (36.9)</td>
</tr>
<tr>
<td>Lower chest wall indrawing</td>
<td>64 (57.7)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Respiratory rate</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>02-11 months&gt;50</td>
<td>53 (47.7)</td>
</tr>
<tr>
<td>&gt;012 months&gt;40</td>
<td>52 (46.8)</td>
</tr>
<tr>
<td>No pneumonia</td>
<td>6 (5.4)</td>
</tr>
</tbody>
</table>

<p>| | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Stiff neck</td>
<td>9 (8.1)</td>
</tr>
<tr>
<td>Oedema</td>
<td>6 (5.4)</td>
</tr>
<tr>
<td>Pallor</td>
<td>31 (27.9)</td>
</tr>
</tbody>
</table>
4.2 Blood glucose levels

Four (4) out of the 111 children who took part in the study were found to be hypoglycemic giving a prevalence of 3.6%. 102 (91.9%) children had normal blood glucose levels while 5(4.5%) children were hyperglycaemic. The mean blood glucose was 6.7mmol/ltr. The results are shown in Table 2 and 3 as well as Figure 1 below.

Table 2: Prevalence of hypoglycemia

<table>
<thead>
<tr>
<th>Variable</th>
<th>Point estimate</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean blood glucose</td>
<td>6.7</td>
<td>6.3, 7.1</td>
</tr>
<tr>
<td>Hypoglycemia, n (%)</td>
<td>4 (3.6)</td>
<td>0.9, 7.2</td>
</tr>
</tbody>
</table>

Table 3: Blood glucose levels

<table>
<thead>
<tr>
<th>Variable</th>
<th>Frequency (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypoglycemia</td>
<td>4 (3.6)</td>
</tr>
<tr>
<td>Normoglycemia</td>
<td>102 (91.9)</td>
</tr>
<tr>
<td>Hyperglycemia</td>
<td>5 (4.5)</td>
</tr>
</tbody>
</table>

Figure 1: Blood glucose levels
4.3 Assessment of associated factors

Sixty nine children (62.2%) out of the 111 children recruited into the study had been unwell for less than a week prior to arrival at the hospital while the remaining 42 (37.8%) were found to have been unwell for more than a week. The majority of the children (76.6%) had their last meal within 12 hours prior to admission. This is illustrated in Figures 2 and 3 below.

![Figure 2: Duration of illness before admission in days](image1)

![Figure 3: Last meal](image2)
Table 4 below shows the medications that were administered prior to arrival at the hospital. 59.5% and 60.4% of the children were on antibiotics and analgesics respectively before arrival at the hospital. 8 children (7.2%) had been given antimalarials while 12 (10.8%) children and 6 (5.4%) children were on antihistamines and cough syrups respectively on arrival at the hospital. 2 (1.8%) children received traditional medicines prior to admission.

Table 4: Medications given before admission

<table>
<thead>
<tr>
<th>Variable</th>
<th>Frequency (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Antibiotics</td>
<td>66 (59.5)</td>
</tr>
<tr>
<td>Antimalarials</td>
<td>8 (7.2)</td>
</tr>
<tr>
<td>Analgesics</td>
<td>67 (60.4)</td>
</tr>
<tr>
<td>Antihistamines</td>
<td>12 (10.8)</td>
</tr>
<tr>
<td>Cough syrups</td>
<td>6 (5.4)</td>
</tr>
<tr>
<td>Traditional medicine</td>
<td>2 (1.8)</td>
</tr>
</tbody>
</table>
4.4 Primary clinical diagnosis

The primary clinical diagnoses made by the attending doctor after history taking and physical examination are shown in Table 5 and Figure 4 below. Out of the 8 (7.2%) suspected cases of malaria, 5 children (4.5%) had a positive malaria slide. 44 (39.6%) were diagnosed to have pneumonia, 18 (16.2%) had severe malnutrition, 20 (18%) were diagnosed with meningitis and 30 (27%) with gastroenteritis.

Table 5: Primary clinical diagnosis

<table>
<thead>
<tr>
<th>Variable</th>
<th>Frequency (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Malaria parasite</td>
<td></td>
</tr>
<tr>
<td>Positive</td>
<td>5 (4.5)</td>
</tr>
<tr>
<td>Negative</td>
<td>106 (95.5)</td>
</tr>
<tr>
<td>Malaria</td>
<td>8 (7.2)</td>
</tr>
<tr>
<td>Pneumonia</td>
<td>44 (39.6)</td>
</tr>
<tr>
<td>Severe Malnutrition</td>
<td>18 (16.2)</td>
</tr>
<tr>
<td>Meningitis</td>
<td>20 (18.0)</td>
</tr>
<tr>
<td>Gastroenteritis</td>
<td>30 (27.0)</td>
</tr>
</tbody>
</table>

Figure 4: Primary clinical diagnosis
4.5 Outcome at 24 hours of admission

As shown in Table 6 below, of all the children recruited into the study and subsequently admitted, only one child (0.9%) succumbed within 24 hours of admission. All the other children (99.1%) were alive 24 hours after admission.

Table 6: Outcome at 24 hours of admission

<table>
<thead>
<tr>
<th>Variable</th>
<th>Frequency (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Alive</td>
<td>110 (99.1)</td>
</tr>
<tr>
<td>Dead</td>
<td>1 (0.9)</td>
</tr>
</tbody>
</table>

4.6 Factors associated with hypoglycemia

In this study, none of the factors assessed was significantly associated with the occurrence of hypoglycaemia as can be seen in Table 7 below.

Table 7: Lack of association between hypoglycemia and potential associated factors

<table>
<thead>
<tr>
<th>Variable</th>
<th>Hypoglycemia n (%)</th>
<th>Normal n (%)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Last meal</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;012</td>
<td>3 (75.0%)</td>
<td>77 (75.5%)</td>
<td>1.000</td>
</tr>
<tr>
<td>&gt;012</td>
<td>1 (25.0%)</td>
<td>25 (24.5%)</td>
<td></td>
</tr>
<tr>
<td>WHZ score</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Appropriate</td>
<td>2 (50.0%)</td>
<td>32 (32.0%)</td>
<td>0.341</td>
</tr>
<tr>
<td>-1SD</td>
<td>0 (0.0%)</td>
<td>24 (24.0%)</td>
<td></td>
</tr>
<tr>
<td>-2SD</td>
<td>1 (25.0%)</td>
<td>26 (26.0%)</td>
<td></td>
</tr>
<tr>
<td>-3SD</td>
<td>0 (0.0%)</td>
<td>13 (13.0%)</td>
<td></td>
</tr>
<tr>
<td>-4SD</td>
<td>1 (25.0%)</td>
<td>5 (5.0%)</td>
<td></td>
</tr>
<tr>
<td>Temperature</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>035-36.5</td>
<td>0 (0.0%)</td>
<td>19 (18.8%)</td>
<td>0.166</td>
</tr>
<tr>
<td>36.6-37.5</td>
<td>2 (50.0%)</td>
<td>51 (50.5%)</td>
<td></td>
</tr>
<tr>
<td>38.6-40</td>
<td>1 (25.0%)</td>
<td>29 (28.7%)</td>
<td></td>
</tr>
<tr>
<td>&gt;040</td>
<td>1 (25.0%)</td>
<td>2 (2.0%)</td>
<td></td>
</tr>
<tr>
<td>Level of consciousness</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>A</td>
<td>3 (75.0%)</td>
<td>76 (74.5%)</td>
<td>1.000</td>
</tr>
<tr>
<td>V</td>
<td>0 (0.0%)</td>
<td>5 (4.9%)</td>
<td></td>
</tr>
<tr>
<td>P</td>
<td>1 (25.0%)</td>
<td>18 (17.6%)</td>
<td></td>
</tr>
<tr>
<td>U</td>
<td>0 (0.0%)</td>
<td>3 (2.9%)</td>
<td></td>
</tr>
<tr>
<td>Dehydration</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No dehydration</td>
<td>2 (50.0%)</td>
<td>60 (58.8%)</td>
<td>0.483</td>
</tr>
<tr>
<td>Condition</td>
<td>Yes</td>
<td>No</td>
<td>Total</td>
</tr>
<tr>
<td>-----------------------------------</td>
<td>-----------</td>
<td>-----------</td>
<td>-----------</td>
</tr>
<tr>
<td>Some dehydration</td>
<td>0 (0.0%)</td>
<td>2 (50.0%)</td>
<td>14 (13.7%)</td>
</tr>
<tr>
<td>Severe dehydration</td>
<td>0 (0.0%)</td>
<td>21 (20.6%)</td>
<td>7 (6.9%)</td>
</tr>
<tr>
<td>Shock</td>
<td>3 (75.0%)</td>
<td>1 (25.0%)</td>
<td>72 (70.6%)</td>
</tr>
<tr>
<td>Unable to drink or breastfeed</td>
<td>2 (50.0%)</td>
<td>38 (37.3%)</td>
<td>64 (62.7%)</td>
</tr>
<tr>
<td>Difficulty in breathing</td>
<td>1 (25.0%)</td>
<td>93 (91.2%)</td>
<td>0.000</td>
</tr>
<tr>
<td>Lower chest wall indrawing</td>
<td>2 (50.0%)</td>
<td>57 (55.9%)</td>
<td>0.000</td>
</tr>
<tr>
<td>Respiratory rate 02-11months&gt;50</td>
<td>3 (75.0%)</td>
<td>46 (47.9%)</td>
<td>0.000</td>
</tr>
<tr>
<td>Respiratory rate &gt;012months&gt;40</td>
<td>1 (25.0%)</td>
<td>50 (52.1%)</td>
<td>0.000</td>
</tr>
<tr>
<td>Neck stiffness</td>
<td>0 (0.0%)</td>
<td>9 (8.8%)</td>
<td>0.000</td>
</tr>
<tr>
<td>Oedema</td>
<td>0 (0.0%)</td>
<td>4 (100.0%)</td>
<td>0.000</td>
</tr>
<tr>
<td>Pallor</td>
<td>0 (0.0%)</td>
<td>29 (28.4%)</td>
<td>0.000</td>
</tr>
<tr>
<td>Antimalarials</td>
<td>0 (0.0%)</td>
<td>73 (71.6%)</td>
<td>0.000</td>
</tr>
<tr>
<td>Malaria parasite</td>
<td>1 (25.0%)</td>
<td>3 (2.9%)</td>
<td>0.000</td>
</tr>
<tr>
<td>Malaria</td>
<td>3 (75.0%)</td>
<td>99 (97.1%)</td>
<td>0.000</td>
</tr>
<tr>
<td>Malnutrition</td>
<td>0 (0.0%)</td>
<td>8 (7.5%)</td>
<td>0.000</td>
</tr>
<tr>
<td>Pneumonia</td>
<td>2 (50.0%)</td>
<td>42 (39.3%)</td>
<td>0.000</td>
</tr>
<tr>
<td>Meningitis</td>
<td>1 (25.0%)</td>
<td>18 (17.6%)</td>
<td>0.000</td>
</tr>
<tr>
<td>Gastroenteritis</td>
<td>2 (50.0%)</td>
<td>20 (18.7%)</td>
<td>0.000</td>
</tr>
</tbody>
</table>
4.7 Hypoglycemia and its effect on the outcome at 24 hours of admission

The presence of hypoglycaemia was not significantly associated with the outcome at 24 hours of admission as shown in Table 8 below.

Table 8: Effect of hypoglycemia on the outcome at 24 hours of admission

<table>
<thead>
<tr>
<th>Variable</th>
<th>Hypoglycemia n (%)</th>
<th>Normoglycemic n (%)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Outcome</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Alive</td>
<td>4 (100.0%)</td>
<td>106 (99.1%)</td>
<td>1.000</td>
</tr>
<tr>
<td>Dead</td>
<td>0 (0.0%)</td>
<td>1 (0.9%)</td>
<td></td>
</tr>
</tbody>
</table>
5.0 DISCUSSION

Hypoglycemia is not a disease-specific symptom but may be regarded as a serious metabolic complication in acute severe infections in young children. It is an independent risk factor for death in severe childhood infections in the tropics.

In this study, the prevalence of hypoglycemia was found to be 3.6%; with a 95% C.I (0.9, 7.2). The cut off plasma glucose level used in this study was 2.5mmol/litre in sick children and 3.0mmol/litre in severely malnourished children. Studies from other developing countries such as a study by Elusiyan et al (17) in Nigeria found a prevalence of 6.4% while Solomon and colleagues (15) reported a prevalence of hypoglycemia of 7.1% among Mozambican children. Osier (13) reported a prevalence of 7.3% among paediatric emergency admissions at a Kenyan rural district hospital. A study in the United States by Pershad et al (18) reported a prevalence of 6.54% of 100000 visits among patients seeking care in their emergency department. Our prevalence of hypoglycemia of 3.6% among sick children at KNH is much lower than the prevalences reported from other studies (13, 15, 17, 18) probably because of our smaller sample size and excluding infants below two months of age.

Children have a limited tolerance to fasting because they have less glycogen stores. In this study, only one child (25%) had their last meal over 12 hours prior to admission but this was not found to have a statistically significant association with the presence or absence of hypoglycemia. This was not in keeping with the findings of other workers (13, 15, 17) who reported significant associations between the interval of the last meal and presence or absence of hypoglycemia. Elusiyan et al (17) found that patients presenting more than 12 hours of their last meal were more likely to be hypoglycemic (p<0.001) while Osier and others (13) reported a prevalence of 20.8% with a strong association to hypoglycemia (O.R =2.1; 95%CI=1.4-3.1, p<0.001).

There was no observed statistically significant association between age, sex and duration of illness and hypoglycemia in the present study. This was in agreement with the findings of other workers (13, 17). The Nigerian study (17), however, found an increasing prevalence of hypoglycemia in children who had a longer duration of illness before admission. Osier et al (13) found the highest prevalence of hypoglycemia among neonates (23%) and children aged between two and four years.
Nutritional status was not found to have a statistically significant association with hypoglycemia in this study as well as among Nigerian children (17) but was significantly associated with hypoglycemia in Kenyan children (13) with a WAZ<3 (p=0.023). One would generally expect a significant relationship between nutritional status and hypoglycemia. The reason why this is so is not clear but it is thought that the hormonal compensatory interplay prevents the occurrence of hypoglycemia.

There was a statistically significant association between the coma score of patients and the presence of hypoglycemia in work done by Elusiyan et al (17), (p<0.01) and Osier et al (13), (p<0.001) who reported a mortality rate of 31.8%. The present study found a prevalence of hypoglycemia of 25% in children with a reduced level of consciousness but this was not found to be statistically significant.

This study has shown that hypoglycemia can complicate many childhood diseases. This is in agreement with the findings of other workers (13, 15-17, 22-25). Out of the four hypoglycemic cases, one child (25%) was diagnosed with malaria and had a positive blood slide for malaria parasites. Elusiyan et al (17) reported malaria as the leading cause of hypoglycemia in Nigerian children at 44%, followed by 31% in Gambian children (24) and 20% in Malawian children (25). This was high compared to 8.4% reported by Osier et al (13), 16% reported by English et al (16) and 5.2% reported in Mozambican children (15). A frequency of hypoglycemia of 11.6% was found in Togo children (22) and 11.4% in Ugandan children (23) with severe malaria. The impairment in hepatic gluconeogenesis has been considered to be the most likely mechanism in the causation of hypoglycemia especially if there is acidosis and renal impairment (16). The Kenyan study (13) also found a strong association between hypoglycemia and a positive malaria slide (p=0.006).

Hypoglycemia has also been associated with severe malnutrition. 25% of the hypoglycemic cases in the present study had severe malnutrition but this was not statistically significant. Osier et al (13), on the other hand, found a strong association between severe malnutrition and hypoglycemia (p=0.023) with a prevalence of 6.6%. However, there was no evidence of association of hypoglycemia with mortality in children with malnutrition. This was in contrast to the Nigerian study (17) where there was 100% mortality of the patients who had protein energy malnutrition, specifically Kwashiokor, as well as hypoglycemia. Huq et al (21) did not find any
significance between hypoglycemia and malnutrition. One of the reasons could be hormonal compensatory interplay preventing hypoglycemia.

Diarrhoeal illness has been associated with hypoglycemia. In this study, 25% of the hypoglycemic cases had diarrhoeal disease. This prevalence was high when compared to other studies, possibly due to the lower sample size. Huq et al (21) enrolled 782 under-5 children with diarrhoea to determine the prevalence of hypoglycemia which he found to be 11%. In his study, hypoglycemia was strongly associated with bacteremia (p=0.026). Among the hypoglycemic children, 11% were found to have bacteremia compared to 5% of the non-hypoglycemic children. Logistic regression in his study found that bacteremic children with clinical sepsis were four times more likely to develop hypoglycemia (OR=4.2, 95% CI=1.4-12.9, p=0.012). Osier et al (13) found a prevalence of 5.5% while Solomon et al (15) found a prevalence of 7.1% among Mozambican children with diarrhoeal disease. Nyikuri et al (33) found a prevalence of hypoglycemia of 4.2% in children admitted with diarrhoeal disease at Kenyatta National Hospital. Contrary to the studies mentioned above where hypoglycemia was found to be associated with diarrhoeal diseases, hypoglycemia was not found in any of the patients admitted for gastroenteritis in the Nigerian study (17).

Pneumonia has been found to be the leading cause of hypoglycemia in this study with a prevalence of 50% although there was no statistically significant association between the two. The only child who succumbed in this study within 24 hours of admission had pneumonia and was normoglycemic. A prevalence of 21% was reported among Mozambican children (15) and pneumonia was the leading diagnosis associated with hypoglycemia in that study while Elusiyan et al (17) reported a prevalence of 8.6%. Elusiyan attributed the low prevalence in his study to be due to the high usage of antibiotics for upper respiratory tract infections thereby preventing its spread or progression to the lower respiratory tract.

Hypoglycemia is a serious complication of malaria and other infectious diseases and markedly increases mortality rates. Contrary to the findings in previous studies (13, 15-17, 21, 29), none of the children found to be hypoglycemic in this study succumbed to their illness. This may be attributed to the small sample size. This study also looked at mortality within the first 24 hours of admission unlike other mortality audits in KNH which are conducted over a longer period of time with recruitment of a wider age group. Most deaths occur after 48 hours and even more 7
days after admission. The presence of hypoglycemia at admission has been found to be significantly associated with death and dying within 24 hours of admission (17). It is also possible that due to the ETAT training that almost all residents undergo before being posted to clinical areas has led to stabilization of the sick children especially within the first 24 hours hence the lower mortality rate during this period. There is need, however, for larger studies on hypoglycemia as a cause of death in children admitted to KNH.

Elusiyan et al (17) in 2006 reported the highest mortality in hypoglycemic children at 28.6% compared to other studies, against 4.2% mortality in normoglycemic children (p<0.01). This was followed by Huq et al (21) who reported a mortality rate of 28% in hypoglycemia compared to 14% in normoglycemia (OR 2.4), English et al (16) reported a mortality rate of 28% compared to 7% in normoglycemic children (p=0.003), while Osier et al (13) reported a mortality rate of 20.2% compared to 3.8% mortality in normoglycemic children (p<0.001). Solomon et al (15) reported a mortality rate of 16.3% in hypoglycemic cases compared to 3.2% in normoglycemic children (RR-5.8) while Schellenberg et al (29) reported a mortality rate of 12.4% compared to 3% in normoglycemic children (OR 6.7).

In conclusion, hypoglycemia is a serious complication of infectious diseases and is not itself a disease-specific symptom. All emergency admissions should be screened for hypoglycemia and aggressively managed if present. Presumptive treatment is recommended for severely ill children where diagnostic facilities are lacking.
6.0 CONCLUSIONS
The prevalence of hypoglycaemia among sick children aged 2-60 months admitted to KNH was found to be 3.6%.
None of the factors studied were found to be significantly associated with hypoglycaemia.
Hypoglycaemia did not have an effect on the outcome at 24 hours of admission in this study.

7.0 STUDY LIMITATIONS
Some children had more than one co-morbidity at admission therefore relating hypoglycemia with the primary diagnosis proved to be a challenge.

The number of patients found to have hypoglycemia was small therefore analysis was limited.

8.0 RECOMMENDATIONS
There is need for further, larger studies on the prevalence of hypoglycaemia and the long term outcomes of hypoglycaemia in children.
APPENDIX I: QUESTIONNAIRE

Study Identification Number__________ Date of Interview______________

Biodata

1. Age of the patient

    Years __________ Months_____________

    Date of Birth____________________

2. Sex

    a) Male ________ b) Female ________

Assessment of risk factors

3. Duration of illness before admission (days).

    a) <1 __________

    b) 1-7__________

    c) 7-30_________

    d) >30__________

4. Last meal

    a) <12 hours_______

    b) >12 hours_______
5. Symptoms

<table>
<thead>
<tr>
<th>i) Present</th>
<th>ii) Absent</th>
</tr>
</thead>
<tbody>
<tr>
<td>a) Fever</td>
<td></td>
</tr>
<tr>
<td>b) Cough</td>
<td></td>
</tr>
<tr>
<td>c) Difficulty in breathing</td>
<td></td>
</tr>
<tr>
<td>d) Convulsions</td>
<td></td>
</tr>
<tr>
<td>e) Vomiting</td>
<td></td>
</tr>
<tr>
<td>f) Diarrhoea</td>
<td></td>
</tr>
<tr>
<td>g) Others (specify)</td>
<td></td>
</tr>
</tbody>
</table>

6. Medication given before admission:

<table>
<thead>
<tr>
<th>i) Present</th>
<th>ii) Absent</th>
</tr>
</thead>
<tbody>
<tr>
<td>a) Antibiotics</td>
<td></td>
</tr>
<tr>
<td>b) Antimalarials</td>
<td></td>
</tr>
<tr>
<td>c) Analgesics</td>
<td></td>
</tr>
<tr>
<td>d) Antihistamines</td>
<td></td>
</tr>
<tr>
<td>e) Cough syrups</td>
<td></td>
</tr>
<tr>
<td>f) Traditional medicine</td>
<td></td>
</tr>
<tr>
<td>g) Others (specify)</td>
<td></td>
</tr>
</tbody>
</table>
APPENDIX II: CLINICAL BIODATA FORM

Study Number_________ Date of admission________________ Time of admission______
I.P number______________ Ward_______

Biodata

1. Sex

   a) Male _______ b) Female _______

2. Age

   Years ________ Months ________

Assessment of Risk Factors


   Weight (kg) ______________ Height (cm) ______________

   Z- Score (weight for length/height) a) appropriate ______

   b) -1SD____

   c) -2SD____

   d) -3SD____

   e) -4SD____

4. Observations

   a) Temperature at admission (°C)

      i) 35.0-36.5________

      ii) 36.6-37.5________
iii) 37.6-38.5__________

iv) 38.6-40__________

v) >40_______________

b) Blood glucose level (mmol/L) ____________

c) Blood slide for malaria parasites i) positive _____ ii) negative______

5. Examination

a) Level of consciousness: i) A ii) V iii) P iv) U

b) Dehydration:
   i) No dehydration ______________
   ii) Some dehydration ______________
   iii) Severe dehydration ______________
   iv) Shock ______________

c) Able to drink/breastfeed i) Yes_______ ii) No________

d) Deep breathing _______ _________

e) Lower chest wall indrawing _______ _________

f) Respiratory rate:
   i) 2-11 months ≥50_________ ii) >12 months ≥40___________

g) Stiff neck i)Yes_______ ii)No________

h) Oedema _______ _________

i) Pallor _______ _________

33
Assessment of the outcome at 24 hours of admission

Outcome of admission

a) Alive _____________
b) Dead _____________

Primary clinical diagnosis (by the attending doctor)

a) Malaria ______________
b) Pneumonia ______________
c) Gastroenteritis __________
d) Malnutrition ____________
e) Meningitis ______________
f) Others (specify)____________
g) Missing ________________
APPENDIX III: CONSENT FORM FOR PARTICIPATION IN THE STUDY

Study Identification Number: ____________ Date: ____________________________

Study title:

Prevalence of hypoglycemia among children admitted at Kenyatta National Hospital (KNH).

Investigator: Dr Joan Ong’are (MBChB)
Paediatric Registrar, University of Nairobi
Tel: 0722237083

Supervisors: Dr Daniel Njai
Senior Lecturer, Department of Paediatrics and Child health
University of Nairobi, Tel 0722682929
Dr Ahmed Laving
Lecturer, Gastroenterologist
Department of Paediatrics and Child health, University of Nairobi
Tel: 0724644122

Investigator’s statement

My name is Dr Joan Ong’are; I am a postgraduate student in the Department of Paediatrics and Child Health at the University of Nairobi undertaking a study to determine the number of children admitted who have low blood sugar. We request permission for you and your child to participate in this study. The consent form provided is to allow you to make an informed decision on whether or not to participate in the study. Please read this form carefully, asking any questions or seeking any clarifications from the information provided.
**Brief description of the Study**

Hypoglycemia or low blood sugar is a common metabolic abnormality in childhood that can be treated immediately once diagnosed. If left untreated, it can cause brain damage and even death. Hypoglycemia is also associated with childhood diseases and malnutrition. It worsens these conditions and puts the affected children at a higher risk of death. All sick children should therefore have their blood sugar taken and if low, treated accordingly. This study will help us know how often this condition occurs in sick children admitted to KNH.

**Benefits**

The results of the study will be discussed with you and the baby’s doctor. If your baby is found to have any abnormality in the blood glucose levels, the doctor caring for the baby will immediately be informed and treatment given. The results will help us know the burden of the condition and help health workers in this facility and beyond to improve care given to all sick children. The results will also inform caregivers of the risk factors that can lead to low blood sugars and the dangers of a child having low blood sugar levels.

**Risks**

The study will involve pricking the foot of the child to obtain a very small amount of blood (5 micro litres equal to a very small drop of blood) in order to measure blood glucose level. The quantity of blood required is not great enough to lower the child’s haemoglobin levels. Extreme care will be taken while sampling to ensure the highest possible standards of cleanliness are followed.

**Voluntariness**

Participation in this study is completely voluntary. Your baby will not be included in the study without your informed and written consent. There will be no financial rewards for participation in the study and no added expenses will be incurred for participating in the study. The care of your baby will not be compromised in any way as a result of refusal to participate.
Confidentiality

Information obtained about you and your child will be kept strictly confidential. In the event that your child is found to have abnormal blood glucose levels, it will be communicated to the child’s doctor and nursing staff for immediate intervention to be undertaken. Overall findings of this study will be discussed in the Department of Paediatrics and Child Health, University of Nairobi, however, we will not reveal the identity of you and your child during the discussion.

Study Procedure

The study will be conducted as follows: on receiving informed written consent, blood will be obtained from a prick on the underside of the child’s foot. The amount of blood required is very minimal (five micro litres) and all measures will be taken to ensure complete safety of the baby. Abnormal blood glucose if found will communicated to the attending doctor for immediate correction. You will then be assisted to fill in a questionnaire after which your child will be examined.

Problems or Questions

If you have any questions about the study or the use of the results, you can contact the principal investigator: Dr Joan Ong’are by calling 0722 237-083.

If you have any questions regarding your rights as a research participant, you can contact the Kenyatta National Hospital Ethics and Research Committee (KNH-ERSC) by calling 2726300 Ext 44355.
**Consent Form: Participant’s Statement:**

I ____________________________ having received adequate information regarding the research study, risks, benefits hereby AGREE/ DISAGREE (Cross out as appropriate) to participate in the study with my child. I understand that our participation is fully voluntary and that I am free to withdraw at anytime. I have been given adequate opportunity to ask questions and seek clarification on the study and these have been addressed satisfactorily.

Parent’s Signature: ___________________________ Date ___________________

I ____________________________ declare that I have adequately explained to the above participant, the study procedure, risks, benefits and given him/ her time to ask questions and seek clarification regarding the study. I have answered all the questions raised to the best of my ability.

Interviewer’s Signature ___________________________ Date ___________________
APPENDIX IV: KIBALI CHA KUSHIRIKI KWA MTOTO


Ninapeana ruhusa kwa mtoto wangu kushiriki kwa utafiti huu.

Jina .......................................................... Sahihi ..........................................................

Tarehe .......................................................... Saa ..........................................................

Nimeeleza mshiriki kuhusu utafiti huu vile ipasavyo.

Mtafiti .......................................................... Sahihi ..........................................................

Tarehe .......................................................... Saa ..........................................................
APPENDIX V: TIME FRAME

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<thead>
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<th>Number</th>
<th>Activity</th>
<th>Estimated Time</th>
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<tr>
<td>1</td>
<td>Proposal Development and Presentation</td>
<td>Dec 2011 to Jan 2012</td>
</tr>
<tr>
<td>2</td>
<td>Submission of proposal for ethical approval</td>
<td>Feb 2012</td>
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<tr>
<td>3</td>
<td>Pretesting and seeking permission</td>
<td>May to June 2012</td>
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<td>4</td>
<td>Data Collection</td>
<td>July to September 2012</td>
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<td>5</td>
<td>Data Analysis</td>
<td>October to November 2012</td>
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<tr>
<td>6</td>
<td>Thesis writing</td>
<td>December 2012</td>
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<tr>
<td>7</td>
<td>Thesis submission</td>
<td>January 2013</td>
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## APPENDIX VI: STUDY REQUIREMENTS AND BUDGETARY ESTIMATES

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<th>Category</th>
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<td>Blood glucose tests</td>
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REFERENCES


Dear Dr. Ongare

Research proposal: “Prevalence of Hypoglycemia among children admitted at Kenyatta National Hospital” (P43/02/2012)

This is to inform you that the KNH/UoN-Ethics & Research Committee (KNH/UoN-ERC) has reviewed and approved your above revised research proposal. The approval periods are 18th July 2012 to 17th July 2013.

This approval is subject to compliance with the following requirements:

a) Only approved documents (informed consents, study instruments, advertising materials etc) will be used.
b) All changes (amendments, deviations, violations etc) are submitted for review and approval by KNH/UoN ERC before implementation.
c) Death and life threatening problems and severe adverse events (SAEs) or unexpected adverse events whether related or unrelated to the study must be reported to the KNH/UoN ERC within 72 hours of notification.
d) Any changes, anticipated or otherwise that may increase the risks or affect safety or welfare of study participants and others or affect the integrity of the research must be reported to KNH/UoN ERC within 72 hours.
e) Submission of a request for renewal of approval at least 60 days prior to expiry of the approval period. (Attach a comprehensive progress report to support the renewal).
f) Clearance for export of biological specimens must be obtained from KNH/UoN-Ethics & Research Committee for each batch of shipment.
g) Submission of an executive summary report within 90 days upon completion of the study. This information will form part of the data base that will be consulted in future when processing related research studies so as to minimize chances of study duplication and/or plagiarism.

For more details consult the KNH/UoN ERC website www.uonbi.ac.ke/activities/KNHUoN

“Protect to Discover”
Yours sincerely

[Signature]

PROF. A.N. GUANTAI
SECRETARY, KNH/UCON-ERC

c.c.

The Deputy Director CS, KNH
The Principal, College of Health Sciences, UoN
The Dean, School of Medicine, UoN
The Chairman, Dept. of Paediatrics & Child Health, UoN
The HOD, Records, KNH
Supervisors: Dr. Daniel Njai, Dept. of Paediatrics & Child Health, UoN
Dr. Ahmed Laving, Dept. of Paediatrics & Child Health, UoN

"Protect to Discover"