A STUDY OF THE PREVALENCE AND CORRELATES OF EARLY ONSET NEONATAL HYPOCALCAEMIA IN TERM NEONATES AT KENYATTA NATIONAL HOSPITAL

A DISSERTATION SUBMITTED IN PART FULFILMENT OF MASTERS OF MEDICINE (MMED) DEGREE IN PAEDIATRICS AND CHILD HEALTH, UNIVERSITY OF NAIROBI

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DECLARATION

This dissertation is my original work, and has not been presented for a degree in any university or published anywhere.

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DEDICATION

This study is especially dedicated to all newborns whose lives we struggle to save and their parents whose anxiety we strive to allay.

ACKNOWLEDGEMENTS

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I thank the Almighty God for His continued grace upon me and the strength to undertake the study.

TABLE OF CONTENTS

LIST OF ABBREVIATIONS

KDHS	Kenya Demographic and Health Survey
KNH	Kenyatta National Hospital
VLBW	Very low birth weight
PTH	Parathyroid hormone
PTHrP	Parathyroid hormone related protein

LIST OF APPENDICES

LIST OF TABLES

LIST OF FIGURES

ABSTRACT

Introduction: Hypocalcaemia is a major concern in the neonatal period, often associated with metabolic disturbances whose consequences are high morbidity and mortality. Neonatal hypocalcaemia correlates to foetal and maternal calcium level as the neonate depends on the mother to supply all the calcium during foetal life and lactation period. Studies have shown that calcium intake in women during pregnancy and lactation can be quite low. This would reflect as low calcium level in neonates. It is, therefore, important to assess serum calcium level in the term neonate and correlate it to maternal serum calcium levels at birth.

Study Design: A hospital based short cohort study was conducted for a period of three months.

Study Setting: Kenyatta National Hospital (KNH) labour ward and the designated postnatal wards.

Study Population: The study subjects included all pregnant women admitted at the Kenyatta National Hospital labour ward and later their new born babies.

Study Procedure: A total of 121 pregnant women and later their newborn babies were recruited for this study. Women were enrolled into study after giving an informed consen. Fisher's formula was used to calculate sample size. Consecutive sampling was applied.

Once a pregnant woman was recruited, a questionnaire was administered, her blood pressure was taken, trousseaus and chovtek's signs were elicited and a blood sample was drawn and analysed for levels of calcium, alkaline phosphatase, phosphates and albumin. 24 hours after delivery the baby was assessed for any signs of seizures, its weight, length and head circumference was taken and blood sample drawn and analysed as that of the mother.

Results: 121 pregnant women with a mean age of 27.5 years and a median parity of 1.0 were recruited.121 neonates with a mean gestational age of 39.3 weeks were also recruited. The prevalence of maternal hypocalcaemia was 29(24 %) whereas neonatal hypocalcaemia was at 26 (21.5%). Hypocalcaemia in the neonate was significantly associated with maternal hypocalcaemia (p 0.013).

Conclusion: Neonatal hypocalcaemia is at 21.5% and it is significantly associated with maternal hypocalcaemia.

Recommendations: Routine maternal calcium supplementation during pregnancy should be implemented to prevent maternal and neonatal hypocalcaemia. Serum calcium analysis during pregnancy and neonatal serum calcium analysis at birth should be undertaken to facilitate appropriate intervention with supplements for those who are not on supplementation. A nutritional interventional study is recommended to elicit more valuable information on the role of diet on calcium levels.

1.0 BACKGROUND

1.1 CALCIUM

Calcium is a divalent cation in the body where 99% of it is in bone, mostly as hydroxyapatite (1). Infants have approximately 400mg/kg of calcium where as adults have 950mg/kg (1). Calcium is involved in several functions in the body including: bone mineralisation, blood coagulation, endocytosis, muscle contraction and neuromuscular transmission (2). Free or ionized calcium makes up less than half of the total serum calcium while the remainder is bound to protein, mostly albumin (2).

Hypocalcemia is a total serum calcium concentration less than 8 mg/dL (2 mmol/L) in term infants or 7 mg/dL (1.75 mmol/L) in preterm infants. It is also defined as an ionized calcium level less than 3.0mg/dl (0.75 mmol/l), depending on the method (type of electrode) used. Hypocalcaemia is a major cause of morbidity and mortality in the neonatal period. A high prevalence is reported in neonates of mothers with diabetes mellitus and in neonates with birth asphyxia. The causes of early onset neonatal hypocalcaemia are known to be maternal hypocalcaemia, prematurity and birth asphyxia. Hypocalcemia occurs in as many as 30% of neonates with very low birth weight (<1500 g) and in as many as 89% of neonates whose gestational age at birth is less than 32 weeks (3).

The contribution of maternal hypocalcaemia is very important especially for the term neonate. Studies in South Africa, Nigeria, India, and in the Middle East have determined that dietary deficiency of calcium in pregnant mothers leads to hypocalcaemia in the neonates at birth and during lactation (4,5,6,7,8). Subsequently, the neonates receive suboptimal supply of calcium

from the mother causing concern in the exclusively breastfeeding neonate who depends on the mother to supply all the calcium needed.

1.2 DAILY REQUIREMENTS

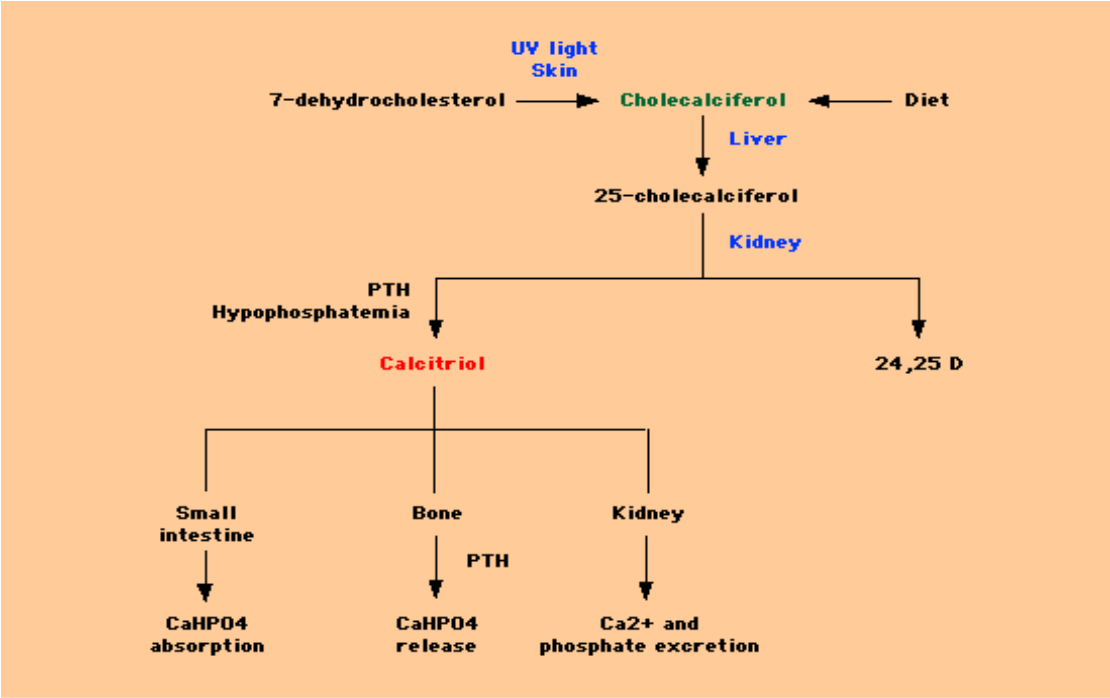
Daily requirements of calcium vary with age. From birth to six months the baby requires 210mg/day, between the ages of seven and twelve months the requirement is 270mg per day and between one to two years the requirement is 500mg per day. The older children of between four to eight years require up to 800mg/day, while adults require up to 1200mg per day (1, 8).

1.3 REGULATION OF CALCIUM

Serum calcium concentration is regulated by a complex hormonal system. Intake is through the gastrointestinal system, losses are through urine and bone is the largest reservoir in the body. A net positive calcium balance is necessary for growth and skeletal mineralization (9).

Calcium regulation is by a feedback mechanism. (Figure1). Parathyroid hormone is stimulated by the presence of low calcium. Parathyroid hormone (PTH) responds by stimulating 1α hydroxylase to produce 1, 25 dihydroxyvitamin D which then stimulates the active transport of calcium across gastrointestinal epithelial cells and bone resorption leading to an increase in serum calcium (9).

FIGURE1: CALCIUM REGULATION



Vitamin D metabolism Metabolic activation of vitamin D to calcitriol and its effects on calcium and phosphate homeostasis. The result is an increase in the serum calcium and phosphate concentrations.

2.0 PATHOPHYSIOLOGY OF EARLY NEONATAL HYPOCALCAEMIA

2.1 ETIOLOGY OF HYPOCALCAEMIA IN THE NEONATE

During the early days of life in normal neonates, the plasma calcium progressively decreases from the relatively high values found in cord blood so that by 24 to 48 hours of life the level is frequently lower than that found in older neonates and infants (10). The decline in plasma calcium in the newborn period tends to be greater in neonates who are not fed or who receive cow's milk than in breastfed neonates (10). The decline is also greatest in neonates who are sick and those born of abnormal pregnancies and labors, including premature neonates, neonates of

diabetic mothers and those with birth asphyxia. In some neonates the plasma calcium level falls to pathologically low levels and tetany or convulsions may result (10).

Several factors have been implicated in the pathogenesis of the hypocalcaemia observed in the early neonatal period, including the dietary intake of calcium and phosphorus, and the immaturity of both the parathyroid and renal function. Refractoriness to parathyroid hormone and hypoparathyroidism together with impaired renal clearance of phosphates have been observed and have all been attributed to immaturity in the neonate (10). In addition, various complications of pregnancy and labour including diabetes mellitus, premature birth, prolonged and difficult labor, and cesarean section have been observed to be of significancy in the syndrome of neonatal hypocalcaemia (10).

Causes of hypocalcaemia in the neonate can be classified according to the age of the neonate. Early neonatal hypocalcaemia occurs between 48 and 72 hours after birth (3). Maternal hypocalcaemia causes foetal hypocalcaemia because of an inadequate transfer of calcium to the foetus (3). In the case of the premature neonate, prematurity leads to poor calcium intake, decreased responsiveness to vitamin D, increased calcitonin, and hypoalbuminemia leading to decreased total but normal ionized calcium (3). Birth asphyxia also plays a role by necessitating delayed introduction of breastfeeding. Diabetes mellitus in the mother leads to hypomagnesaemic state in the foetus which induces functional hypoparathyroidism and hypocalcaemia in the neonate (3). A high incidence of birth asphyxia and prematurity in neonates of diabetic mothers are also contributing factors. Neonates with intra uterine growth restriction may have hypocalcemia in the presence of prematurity and perinatal asphyxia (1,3,10).

Cow's milk contains 1200 to 1790 mg of calcium and 900 to 1960 mg of phosphorus per litre. By contrast, 1 litre of human milk contains150 to 400mg of calcium and 90 to196 mg of phosphorus. Although the absolute amounts of calcium and phosphorus are higher in cow's milk, the calcium phosphorus ratio is lower, averaging 1.3 in cow's milk as compared to 2.2 in breast milk (10). In neonates who are fed cow's milk, the serum phosphorus tends to rise as the calcium decreases owing to this high phosphorus load as compared to breastfed neonates (10).

2.2 EFFECTS OF NEONATAL HYPOCALCAEMIA

The history in neonates with hypocalcaemia varies depending on their age. Newborns might have no symptoms. They may also present with lethargy, a history of poor feeding, vomiting and abdominal distention. Poor muscle tone is responsible for the distension (3).

The hallmark of neonatal hypocalcaemia is neonatal seizures due to central nervous irritation (3). Tetany and other signs of nerve irritability, such as the Chvostek sign, carpopedal spasm, the Trousseau sign, and stridor are commonly encountered. Apnoea and cyanosis due to laryngeal spasms have also been reported (1).

Several studies have indicated that hypocalcaemia might be the cause of rickets in some cases and not vitamin D deficiency. Case reports of rickets among neonates attributable to extremely low dietary calcium intakes were published as early as 1970s (4) . In a separate study in Nigeria, Tom D established that Nigerian children with rickets have a better response to treatment with calcium alone or in combination with vitamin D than treatment with vitamin D alone (5).

2.3 CALCIUM METABOLISM IN PREGNANCY

Calcium metabolism is dramatically altered by pregnancy and lactation. The normal foetal skeleton accumulates about 30g of calcium by the end of pregnancy (at term) (11). This is proportional to the fetal weight. The largest proportion (80%) of that accretion occurs in the third trimester, at a rate of about 250-300 mg/day (11).

Total serum calcium levels fall early in pregnancy, due to haemodilution and the consequent decline in serum albumin. The reference range in pregnancy is therefore slightly lower than in non pregnancy state at 2.0 to 2.40 mmol/l(12). Ionized calcium levels and phosphate levels remain normal throughout pregnancy (11, 13). PTH levels fall to 10-30% of the mean non-pregnant range in the first trimester but increase again to the mid-normal range by term (14,15). Serum calcitonin levels increase during gestation (16), partly due to extrathyroidal synthesis in the placenta and breast. While PTH levels decline, total and free 1,25-dihydroxyvitamin D levels increase 2-fold in the first trimester, then remain constant until term (16,17). The maternal kidneys are the primary source for this increase in vitamin D secondary to up-regulation of the renal 1α-hydroxylase.

There is an increase of 1,25-dihydroxyvitamin D, leading to increased intestinal expression of the vitamin D-dependent calcium binding protein calbindin9K-D (18). This leads to a doubling in intestinal calcium absorption by 12 weeks of gestation (17), and appears to be the major maternal adaptation to supply the fetal calcium requirements. Prolactin and somatomammotropin may also play roles in this increased calcium absorption (18). Animal models suggest that this increased calcium intake is stored in the maternal skeleton until required in the third trimester, but this has not been assessed in humans.

Urinary calcium excretion increases early in gestation secondary to an increased calcium load filtered by the kidneys and the increased glomerular filtration rate of pregnancy. The elevation of calcitonin levels may also contribute to the increased calcium urinary excretion in early pregnancy (19).

In pregnancy, bone turnover is increased but bone mineral content is unchanged. Bone biopsies of women who underwent an elective termination of pregnancy in the first trimester revealed increased bone resorption, with increased resorption surface, increased number of resorption cavities, and decreased osteoid (20,21,22).

2.4 CAUSES OF HYPOCALCAEMIA IN THE PREGNANT WOMAN

Dietary deficiencies of both calcium and vitamin D are ranked as the leading causes of hypocalcaemia in pregnant women, especially in developing countries. Sachan in India found a dietary deficiency of 77% in his study population in Northern India (23). In Egypt 503 pregnant women were studied and 66% had dietary deficiency of calcium (24). In a study published in 2007, Harinarayan found calcium intake in south Indian rural women to be as low as 262mg per day. The Indian council of medical research has recommended an intake of at least 400mg per day in the general population (25).

Pregnancy and lactation places a very high demand for calcium on the mother. The pregnant woman's body provides daily doses of between 50 to 330mg of calcium to support the developing fetal skeleton. Along lactation history stretches the demand for calcium on the mother. In his study, Amotayo in Nigeria established that there was a negative correlation between maternal calcium levels and total duration of breastfeeding ($r = -0.083$) such that the

longer the breastfeeding period, the lower the maternal calcium level (6). These high demand coupled with dietary deficiency predisposes the pregnant woman to hypocalcaemia. This state of hypocalcaemia can further be aggravated by a multiple pregnancy (6).

Causes of hypocalcaemia in the general population also play a role in pregnancy. Endocrinopathies such as hyperparathyroidism have been implicated. Metabolic disturbances including hyperphosphataemia, hypomagnesaemia and hypoalbuminaemia are known to cause hypocalcaemia. Prolonged therapy with certain drugs, (such as phenytoin and phenobarbital anticonvulsants), have been associated with hypocalcaemia (26).

2.5 EFFECTS OF MATERNAL HYPOCALCAEMIA

Maternal hypocalcaemia can have far reaching effects on both the mother and the neonate. It can cause fetal hypocalcaemia because of an inadequate transfer of calcium to the foetus (26). Foetal hypocalcaemia results in foetal hyperparathyroidism with attendant skeletal demineralization, subperiostial bone resorption, osteitis fibrosa cystica and, rarely, death. In his study Maghbooli Z , determined that there was significant correlation between maternal and cord blood serum osteocalcin (A calcium-binding protein in bone) and cross laps (markers of bone degradation products) levels (27). The mean cord blood levels of osteocalcin and cross laps were significantly higher than the maternal serum levels at about 1.59 and 1.62 fold, respectively. Serum calcium levels strongly correlated with osteocalcin and cross laps in mothers ($r = 0.21$, $p = 0.001$ and $r = 0.25$, $p = 0.001$, respectively). A significant direct correlation was observed between maternal and cord blood calcium levels ($r=0.23$, $p=0.002$).

Hypercalcaemia can occur in the neonate as a result of neonatal hyperparathyroidism secondary to maternal hypocalcaemia in utero. In his case report, Ira Sha described a one and a half months old male infant presenting with 5 episodes of apnea followed by cyanosis since day one of life. The mother was treated with calcium supplements for hypocalcaemia during pregnancy. It was established that the neonate had hyperparathyroidism secondary to maternal hypocalcaemia (28).

Hypocalcaemia in pregnancy is also known to induce neonatal hyperthyroidism, hypoinsulinaemia and other metabolic alterations, giving rise to low birth weight (29). Calcium deficiency has been associated with low birth weight in other studies. For example Yang in Taiwan studied the association of very low birth weight with calcium levels in drinking water. He determined that there was significant protective effect of calcium on the risk of delivering a very low birth weight baby (30). This view was supported by Cynthia's study in Canada. In her study, she looked at the association of low milk intake during pregnancy with decreased birth weight. She determined that restricting milk intake lowered infant birth weight in otherwise healthy mothers. Calcium supply in cow milk is at 1200mg per litre (31). Maternal hypocalcaemia can also result in fetal loss as shown in animal study 32. In a local study, Wairumbi, in his 2001 unpublished work , investigated the effects of calcium supplementation in pregnancy at KNH. He followed 126 participants from 29 weeks of gestation to delivery. He found that there were significantly more babies weighing 3000grammes and above in the calcium group (at 78.8%) compared to the placebo group at only 66.6% (p value = 0.25). In addition, the rate of caesarean section was found to be lower in the calcium group (at only 18.2%) compared to the placebo group at 46% (33).

Besides the effects to the pregnancy and the fetus, maternal hypocalcaemia has far reaching effects to the mother herself. Indeed it affects various systems in the pregnant woman's body.

Hypocalcaemia can present as an asymptomatic laboratory finding or as a severe, life-threatening condition. In the setting of acute hypocalcaemia, rapid treatment may be necessary. In contrast, chronic hypocalcaemia may be well tolerated, but treatment is necessary to prevent long-term complications (26).

The hallmark of acute hypocalcaemia is neuromuscular irritability. Patients often complain of numbness and tingling in their fingertips, toes, and the perioral region. Paresthesias of the extremities may occur, along with fatigue and anxiety. Muscle cramps can be very painful and progress to carpal spasm or tetany. In extreme cases of hypocalcaemia, bronchial or laryngeal spasm may occur. Muscle symptoms can be so severe that they present as polymyositis with associated elevated muscle-associated isoenzymes. These symptoms are corrected by calcium replacement. Carpal spasm presents as flexion of the wrist in metacarpal phalangeal joints, extension of the interphalangeal joints, and abduction of the thumb (26).

2.6 PREVALENCE OF MATERNAL AND INFANT HYPOCALCAEMIA

Maternal hypocalcaemia was found to be highest in an Egyptian study at 76.8% (24) compared to other third world countries such as India at 66.4% (36) and Nigeria at 47.2% (6). Overall neonatal hypocalcaemia was highest in a Nigerian study by Amotayo at 61.8%. (6). Binmohanna found a prevalence of 18% in Yemen (34) while Behjati found a prevalence of 33.5% in Iran (35). In the USA, a study by Baum determined the prevalence as 31% (37).

Nationally, Nabakwe, in her 1996 unpublished work, studied neonatal hypocalcaemia in premature neonates at Kenyatta National Hospital and Pumwani Maternity hospital in Nairobi. She found that 31% of the premature neonates at KNH and 33% at PMH had hypocalcaemia (38).

2.7 TREATMENT OF HYPOCALCEMIA

In chronic hypocalcaemia, patients can often tolerate severe hypocalcaemia and remain asymptomatic. The decision to treat is dependent on presenting symptoms, and the severity and rapidity with which hypocalcaemia develops. The patient with acute hypocalcaemia may have symptoms of tetany, seizure, or laryngeal spasm requiring aggressive treatment with intravenous calcium administration (26).

Calcium gluconate is the preferred intravenous calcium type as calcium chloride often causes local irritation. Calcium gluconate contains 90 mg of elemental calcium per 10 mL ampule and usually 1 to 2 ampules (180 mg of elemental calcium) diluted in 50 to 100 mL of 5% dextrose is infused over 10 minutes. This can be repeated until the patient's symptoms have cleared. With persistent hypocalcaemia, administration of dilute calcium solution over longer periods of time may be necessary. The goal should be to raise serum calcium by 2 to 3 mg/dL with the administration of 15 mg/kg of elemental calcium over 4 to 6 hours. Calcium should be maintained in the low-normal range. If possible, oral calcium supplementation should be initiated concurrently with 1 to 2 grams of elemental calcium and if warranted, 1, 25-dihydroxyvitamin D (39).

3.0 STUDY JUSTIFICATION

In Kenya, infant mortality is at 52 per 1000 live births according to the 2008/2009 Kenya demographic and health survey (KDHS) (40). This is quite high and there is need to lower this rate. Neonatal hypocalcaemia contributes to the morbidity and mortality in the neonatal period. High rates of neonatal hypocalcaemia have been reported in the premature neonates but there is very limited data on the prevalence in the term neonate in our set up. This study will therefore shade more light on the prevalence in this group and give important hints to the possible correlates of neonatal hypocalcaemia.

4.0 RESEARCH QUESTION

What is the prevalence and correlates of early onset neonatal hypocalcaemia in the term neonate at Kenyatta National Hospital (KNH)?

4.1 STUDY OBJECTIVES

4.1.1 Primary objective

To determine the prevalence of early onset neonatal hypocalcaemia in term neonates at Kenyatta National Hospital.

4.1.2 Secondary objectives

1) To determine the prevalence of maternal peripartum hypocalcaemia at Kenyatta National Hospital.
2) To determine the correlates of early onset neonatal hypocalcaemia at Kenyatta National Hospital.

5.0 METHODS

5.1 STUDY DESIGN

This was a hospital based short cohort study.

5.2 STUDY SETTING

The study took place at KNH labour ward and the designated postnatal wards. KNH is a National Referral and Teaching Hospital that has been in existence for about 106 years. It is an imposing land mark structure standing on a 45 hectare piece of land with several learning and research institutions on its grounds. Among them is the College of Health Sciences (University of Nairobi); the Kenya Medical Training College; and the Kenya Medical Research Institute and National Laboratory (Ministry of Health).

The hospital has a bed capacity of 1800 in patients with an average bed occupancy rate of over 300%. At any given time the hospital hosts between 2500 and 3000 in-patients averaging 80,000 inpatients and 500,000 outpatients annually. The hospital receives pregnant women with complications from other facilities around Nairobi and countrywide in addition to its own pool of pregnant women from the antenatal clinics. It has 4 maternity wards: GF A, GF B, 1A, 1C and a labour ward. The average delivery rate at the hospital is 400births per month which is quite high. The busy obstetric department is manned by a 240 staff force consisting of 36 doctors and several postgraduate students from the University of Nairobi, 179 nurses and 25 support staff.

5.3 STUDY POPULATION

The study population comprised pregnant women admitted to the Kenyatta National Hospital labour ward and their newborn babies

5.3.1 Inclusion criteria

1. Pregnant women admitted at the KNH labour ward.
2. Newborn babies of the above mothers, between 24 to 72 hours of their delivery.

5.3.2 Exclusion criteria

1. All mothers diagnosed with renal and liver disease in the preceding 3 months
2. All mothers with a positive history of use of anti- tuberculous or anti-epileptic drugs within the preceding three months
3. All newborn babies with congenital abnormalities.
4. All mothers with a history of calcium supplementation during the current pregnancy.

5.4 SAMPLING METHOD

The study applied Consecutive sampling where any pregnant woman who met the inclusion criteria qualified for the study.

5.5 STUDY PROCEDURE

The study assistants approached mothers in labour and introduce themselves. They then assessed the eligibility of the mothers' one at a time. An informed written consent was administered to those who met the inclusion criteria. The study assistant then took a comprehensive medical, drug, and obstetric history including cumulative breastfeeding history from the participant. A

physical examination was done including eliciting of both trousseau's and chvostek's signs. A venous blood sample was then taken from the participant's left arm using a sterile technique. This was collected in a plain red topped specimen bottle labeled appropriately. The relevant sections of the questionnaire were then filled. The study assistant returned 24 to 48hrs after delivery, reviewed the nursing notes, and took the baby's history from the mother. He carried out a physical examination of the baby and recorded his findings. The baby's weight height and head circumference were measured. The baby's venous blood sample was then taken from the left hand dorsum using a sterile broken needle technique. This was collected in a plain red topped specimen bottle labeled appropriately. The relevant section of the questionnaire was then filled.

WEIGHT

A baby scale was used to weigh the babies. Model seca .376 portable electronic weighing scale that has a tare facility and weigh in kilograms to the nearest 5 g up to 7.5 kg was used for weighing the babies. Calibration of the scale was done once a week (41).

WEIGHING

The two assistants weighed and recorded there measurements independently. As one assistant was explaining the procedure to the mother,the other was preparing the baby scale. The baby scale was placed on a flat level surface with no obstructions. With the scale empty the green start button was pressed the word SECA &&& and the figure 0.000 appeared on the display. The scale was then ready for use.The assistant helped the mother undress the baby and placed him or her carefully on the scale and waited for the baby to stop moving. Once the baby stopped moving the hold button was pressed. The display flashed when a stable weight was measured. The

display was then frozen and the baby removed and given to the mother. This weight was recorded. The hold function was timed off by pressing hold key again. 0.000 then appeared.

LENGTH

Length was measured on a harpenden infantometer which has a fixed head board and movable footboard. The infantometer was placed on a raised flat surface. A table that is level and stable was used to avoid causing discomfort. The horizontal board was covered with a thin cloth or a soft paper before the naked baby was placed on it. Diapers were removed because they increase the difficulty of holding the infants legs together and straightening them out. The lead measurer stood on the side to hold down the baby's legs with one hand and move the foot board with the other hand. The assisting measurer stood at the head board and positioned the infant's head. The assisting measurer held the infants head so that the top of the head touched the fixed head board. The head was positioned such that a vertical line from the ear canal to the lower boarder of the eye socket was perpendicular to the horizontal board. This head position is known as the Frankfurt vertical plane.

To keep the infant's head in the correct position the assisting measurer gently cupped her hand over the infant's ears. The mother stood close on the side to measure the infant. The lead measurer positioned the infant so that shoulders and hips were aligned at right angles to the long axis of the body. Gentle pressure was applied on the knees to straighten the legs.

To take the measurements the foot board was positioned gently against the infant's feet. It was ensured that the sole of the feet were flat on the board with toes pointing upwards. The

measurements ware recorded to the last completed 1mm. For example, if the length is between 51.3mm and 51.4 mm51.3 mm was recorded.

HEAD CIRCUMFERENCE

A plastic tape marked in centimeters and millimetres was used to measure head circumference. The infant was held in the assistant's or the mother's laps. The lead study assistant sat by the side of the mother. The study assistant ensured that the side of the tape marked in centimeters was on the outside for reading with the zero end in the inferior position.The tape was looped before slipping it over the head. The tape was then anchored first above the eye brows with the zero point on the side closest to the measurer. At the back of the head the tape was positioned over the furthest protuberance of the skull. The other measurer helped by positioning the tape correctly i.e. level, on the other side of the head. Once the tape was positioned correctly was pulled tight to compress the hair and skin. Care was taken not to pull the tape too tight and cause injury to the new born. The reading was taken to the last completed 1mm and then the tape was removed from the infant's head. The value was then immediately recorded on the corresponding section of the questionnaire. The two assistants took time to independently measure and record each anthropometric measurement without revealing the values obtained to each other till the end of the exercise. The two measures were then compared to ensure that the differences between them fall within maximum allowed differences (7mm for length, 5mm for head circumference and 50g for weight). Any measurement falling outside the maximum allowed difference was repeated by both the assistants up to three readings. Before ending the anthropometry session the questionnaire section for anthropometry was checked for completeness (40).

LABORATORY ANALYSIS

Blood samples collected in plain sample bottles were transported in an ice box to the University of Nairobi Paediatric Department Laboratory at Kenyatta National Hospital. Standardization and quality control at this laboratory is done following the national laboratories protocol which meets international standards. Analysis was done using the Olympus kit. This utilizes a spectrophotometric method using a Lisatrol – 1 series machine. Standardized control samples were run every day before the test samples were run. Mothers' blood and newborns' blood serum levels of Calcium, phosphates, alkaline phosphates and albumin were then measured.

6.0. TIME FRAME

The study took two months from the commencement date.

7.0. ETHICAL CONSIDERATIONS

Approval of the study was sought from the Kenyatta national hospital scientific and ethics committee. Two study assistants were recruited. They underwent training to understand the objectives and procedures of the study. These were qualified clinical officers from the paediatric department. The principle investigator introduced the study assistants to the administration and staff of KNH labor ward and postnatal wards. Consent was sort from the eligible clients before enrolling them in the study. The consent form is in appendix ii.

No harm was intended for the subjects. A prick pain was experienced during the withdrawal of the blood sample. The same was explained to the participants before consenting. The participants were not subjected to any extra cost because of the study.

Women and neonates found to be deficient of calcium were contacted and referred to the appropriate clinics for supplementation and follow up.

8.0 DATA MANAGEMENT AND ANALYSIS

8.1 DATA MANAGEMENT

Data from the field was coded and double entered into a computer database designed using MS-Access application. Data cleaning and validation was performed to achieve a clean dataset that was then exported into a Statistical Package format (SPSS). A clean dataset was stored in a computer hard drive disk ready for analysis. Back up files were stored in a flask disc and a CD, this was done regularly to avoid any loss or tampering. All the questionnaires were stored in a lockable drawer for confidentiality.

8.2 DATA ANALYSIS

Data analysis was conducted using SPSS statistical software. Exploratory data techniques were used at the initial stage of analysis to uncover the structure of data and identify outlier or unusual entered values. Univariate analysis was done where descriptive statistics such as proportions were used to summarize categorical variables and measures of central tendency for continuous variables.

For Bivariate analysis, Pearson's Chi-square test or fisher exact test was used to test for the strength of association between categorical variables. All exposure variables (Independent factors) were associated with the dependent variable (neonatalhypocalcaemia) to determine which ones had significant association. Odds Ratio (OR) and 95% Confidence Interval (CI) were used to estimate the strength of association between independent variables and the dependent

variable. The threshold for statistical significance was set at $\alpha = 0.05$ and a two-sided P- value at 95% confidence intervals (CI) reported for corresponding analysis.

Multivariate analysis was done where all independent variables identified to significantly associate with neonatal hypocalcaemia at bivariate analysis were considered together. This was performed using Binary logistic regression where backward conditional method was specified in order to eliminate confounders and effect modifiers. Adjusted odds Ratios (AOR) with their respective 95% Confidence Interval (CI) were used to estimate the strength of association between the retained independent variables and neonatal hypocalcaemia.

9.0 RESULTS

9.1 CHARACTERISTICS OF THE STUDY POPULATION

9.1.1 Selected demographic characteristics

A total of 121 pregnant women admitted to Kenyatta National Hospital and subsequently their newborn babies were sampled. Socio-demographic and medical history data was collected. The pregnant women were interviewed on their specific nutritional information. Mean age of the pregnant women was 27.5years (SD± 4.6), ranging between 22 and 32 years. There median parity was 1 with a range of 0 – 2 while the mean gestational age of their pregnancies was 39.3 (SD 1.1). Majority of the mothers 62(51.2%) had either a college or university degree as shown in table 1 below:

Table 1: Demographic characteristics of the study participants

Variable	Frequency (%)
Age in years	27.5 (4.6)
Mean (SD)	
Parity	1 (0-2)
Median (IQR)	
Gestation age in weeks	39.3 (1.1)
Mean (SD)	
Level of education	
Primary	6(5.0)
Secondary	53(43.8)
College/university	62(51.2)
Occupation	
Housewife	34(28.1)
Formal employment	33(27.3)
Self employment	44(36.4)
Unemployed	10(8.3)

9.1.2 Medical history of the pregnant women

In the review of the mother's medical history, majority of the mothers 108(89%) spent time basking in the sun,7 mothers (5.8%) reported twitching, 3 had perioral paraesthesia and 2 reported carpal pedal spasms within the preceding 3 months as shown in table 2 below

Table 2: Maternal medical history

Variable	Frequency (%)
Calcium supplement	3 (2.5)
Anticonvulsants in the last 3 months	Nil
Treatment of diabetes	Nil
Treatment of high blood pressure	4 (3.3)
Perioral paresthesia	3 (2.5)
Twitching in the last 3 months	7 (5.8)
Carp pedal spasm	2 (1.7)
Tetany in the last 3 months	1(0.8)
Seizures	Nil
Spent time basking in the sun	108 (89.3)
Less than 1 hour	64 (59.3)
More than 1 hour a day	33 (30.6)
Not stated	11 (10.2)

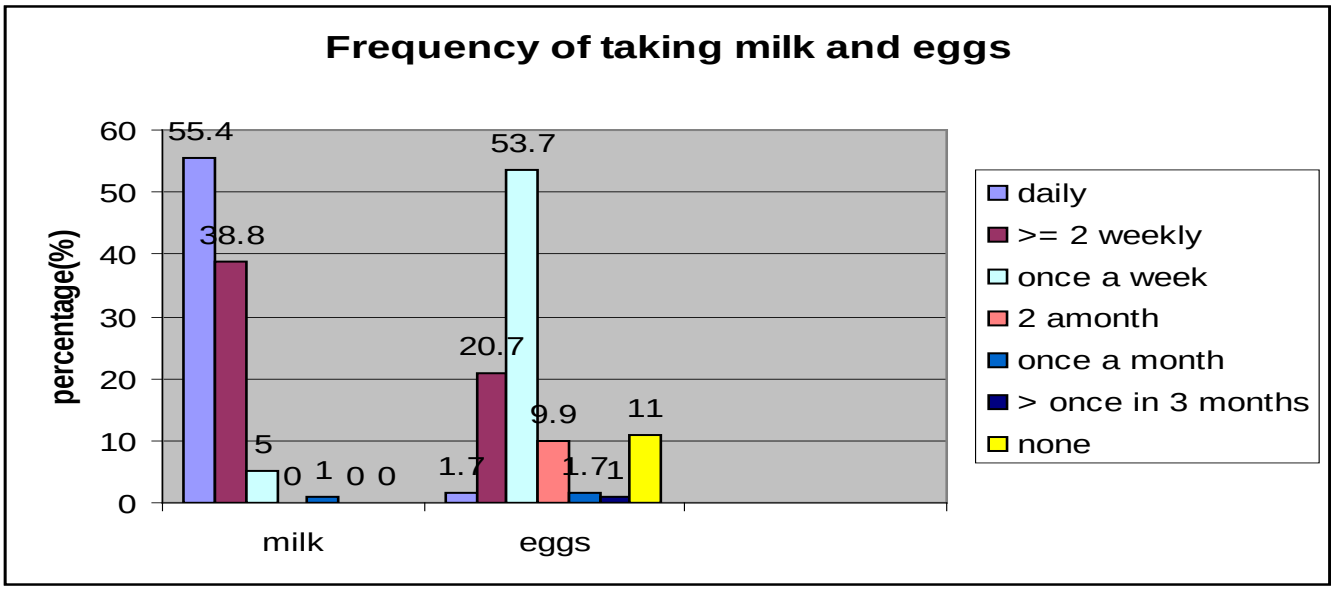
9.1.3 Nutritional characteristics among the pregnant women

Majority of the mothers interviewed 95 (78.5%) had taken milk within the last 24 hours prior to the interview where as only 16 (13.2) had taken eggs within the same period. Indeed 67 (55.4%) reported to have been taking milk on a daily basis as compared to only 2 (1.7%) who took eggs daily. Majority of the mothers 65 (53.7%) preferred to take eggs once a week. 76(62.8%) had taken beans which were mostly boiled without soaking 86 (71.1 %.).Table 3 and figure 2

Table 3: Food taken in the last 24 hrs

Variable	Frequency (%)
Milk	95 (78.5)
Fish	4 (3.3)
Eggs	16 (13.2)
Beans	76(62.8)
Green grams	4 (3.3)
Soya	2 (1.7)
Cereals boiled without soaking	86 (71.1)
Cereals soaked	30 (24.8)
Cereals fermenting	1(0.8)
Cereals sprouting	1 (0.8)

Figure 2: Frequency of taking milk and eggs



9.1.4 Specific clinical findings of the pregnant women

Signs of severe hypocalcaemia (treausseau's sign and chovtek's sign) were not positive in any of the participants. Their mean systolic blood pressure was 109 (SD 9.6) where as the mean diastolic blood pressure was 73.5 (SD 10.5).

9.1.5 Neonatal birth history

The most common mode of delivery was spontaneous vertex delivery (86%). Only 13 babies (0.7%) were delivered via caesarean section.117 (96.7%) babies cried immediately after birth 115 (95%) within the first 5minutes of birth. Majority (95.9%) of the babies were breast fed with only 0.8% of them on infant formula milk. Two babies were reported to have seizures. Other findings were a mean weight of 3kg (SD 0.2), height 47.9 cm (SD 5.4), and a mean head circumference of 34.2cm (SD 3.3). The mean apgar score was 10 (SD 0.2). Table 4

Table 4: Newborns history and clinical findings

Variable	Frequency (%)
Mode of delivery	
Normal	104(86.0)
Cesarean section	13(0.7)
Missing	4 (3.3)
Yes	117(96.7)
Missing	4 (3.3)
Mode of baby feeding	
Breast feeding	116 (95.9)
Infant formula milk	1 (0.8)
Missing	4 (3.3)
Seizures activity	
Yes	2 (1.7)
No	116 (95.9)
Missing	3 (2.5)
Apgar score	10 (0.2)
Body weight	3.0 (0.2)
Height	47.9 (5.4)
Head circumference	34.2 (3.3)

9.1.7 Lab analysis

Both maternal and neonatal blood samples were analysed for serum levels of calcium, phosphates, alkaline phosphates and albumin. The values were as shown in tables 5 and 6 below.

Table 5: lab analysis (neonatal values)

Variable	Mean (SD)/ Median (IQR)
Serum calcium	2.0 (0.7)
Serum phosphate	1.5 (1.0-2.4)
Serum alkaline phosphates	102 (65-102)
Albumin	35.5 (14.6)

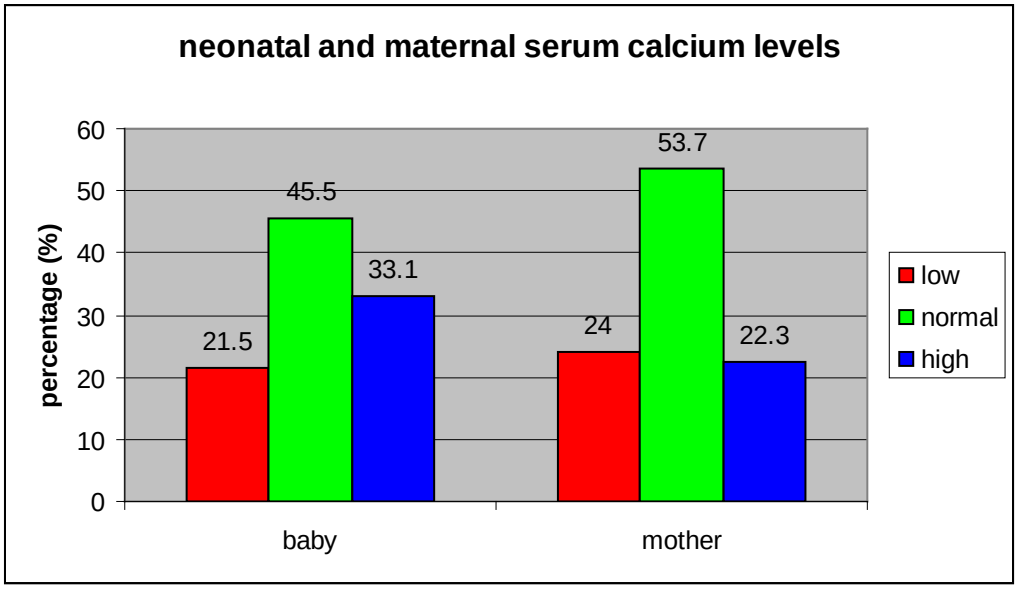
Table 6: Lab analysis (maternal values)

Variable	Mean (SD)/ Median (IQR)
Serum calcium	2.1 (0.4)
Serum phosphate	1.9 (1.2-3.4)
Serum alkaline phosphates	143 (85-143.0)
Albumin	41.7 (13.7)

PREVALENCE OF HYPOCALCAEMIA

Neonatal hypocalcaemia was found in 26(21.5%) of the study population (n = 121) while maternal hypocalcaemia was found in 29(24%) of the study population (, n = 121.) 55 neonates (45.5%) and 65 mothers (53.7%) had normal calcium levels. The remaining 39 neonates (33%) and 27 mothers (22.3%) had hypercalcaemia (Figure 3). Maternal and neonatal hypophosphataemia was found in 6 (5%) and 17 (14%) respectively. High alkaline phosphatase was recorded in 65(53.7 %) of the mothers and30 (24.6%) of the babies (table 7).

Figure 3: Neonatal and maternal serum calcium levels



The neonatal serum calcium level distribution was between 1.3 to 2.7mmol/L (normogram in figure 4) whereas the maternal distribution was between 1.7 and 2.5mmol/L as shown in the normogram in figure 5.

Figure 4: Normogram of neonatal calcium levels

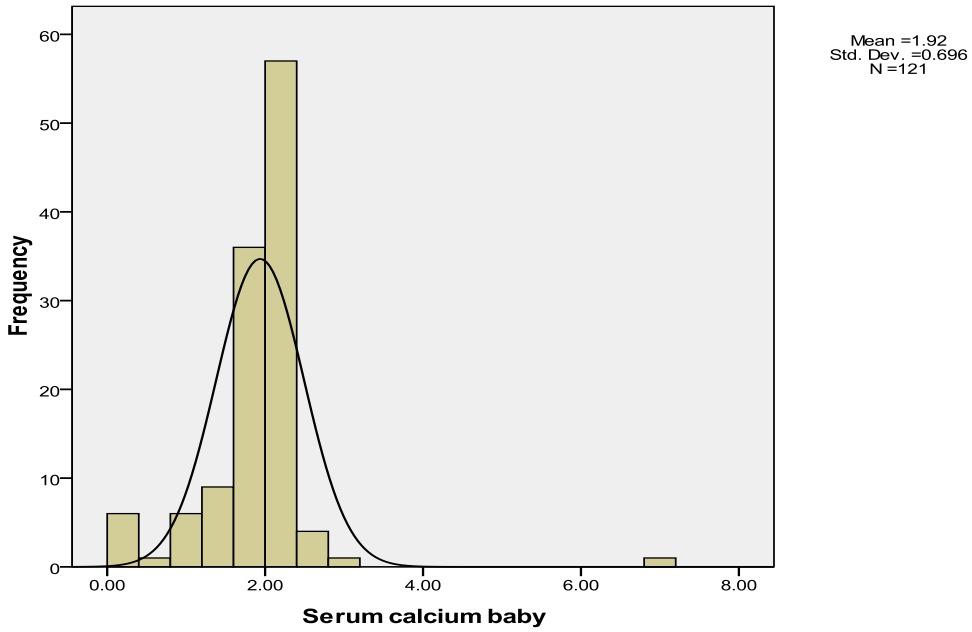


Figure 5: Normogram of maternal calcium levels

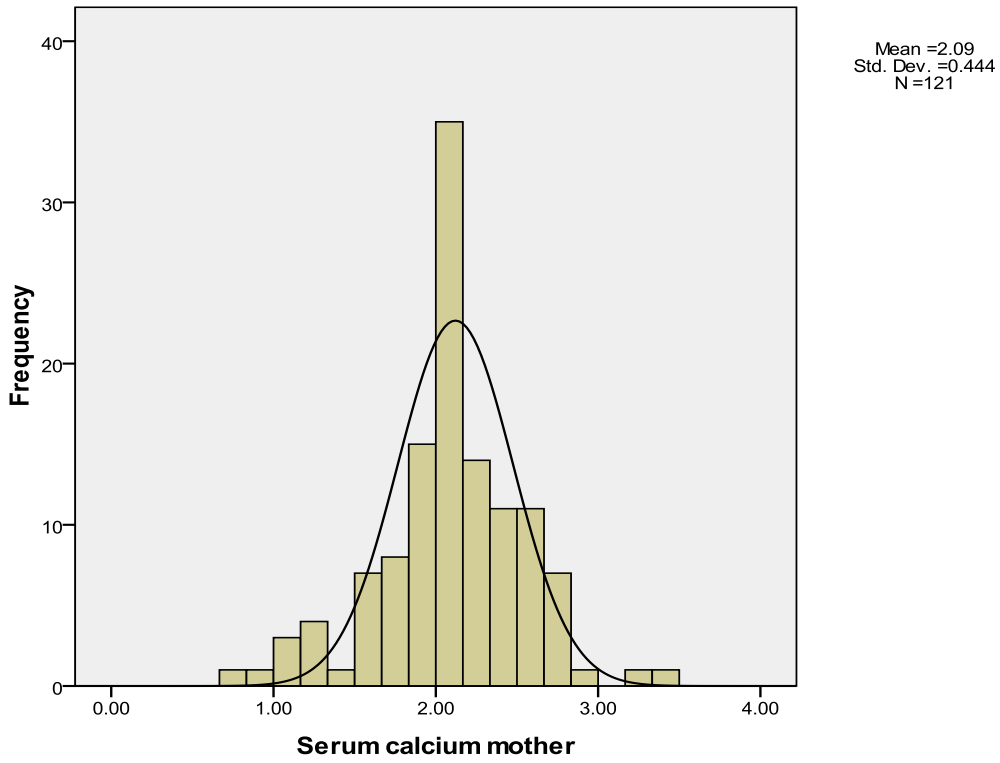


Table 7: Lab results

variable	Mother	Baby
Phosphate	N (%)	N (%)
Low	5 (4.1)	14 (11.6)
Normal	44 (36.4)	57 (47.1)
High	72 (59.5)	50 (41.3)
Alkaline phosphate		
Low	2 (1.7)	11 (9.1)
Normal	54 (44.6)	78 (64.5)
High	65 (53.7)	32 (26.4)
Albumin		
Low	38 (31.4)	58 (47.9)
Normal	67 (55.4)	49 (40.5)
High	15 (12.4)	12 (9.9)
Missing	1 (0.8)	2 (1.7)

ASSOCIATION BETWEEN EARLY ONSET NEONATAL HYPOCALCAEMIA AND SELECTED VARIABLES

Several maternal factors association with neonatal hypocalcaemia was assessed. Maternal hypocalcaemia was significantly associated with neonatal hypocalcaemia. Up to 42.3 % of the babies with hypocalcaemia were born of mothers with low serum calcium levels (p 0.013, 95% CI 3.1). There was no significant association with maternal level of education, parity, cumulative duration of breastfeeding and serum levels of both phosphates and alkaline phosphates as shown in table 8 below.

Table 8: Association between neonatal hypocalcaemia and maternal characteristics

Variable	Calcium level		P value
	Low (n=26)	Normal (n=95)	
Age in years, mean (SD)	27.9 (6.0)	27.4 (4.1)	0.618
Level of education Primary secondary College/university	 1 (16.7%) 11 (20.8%) 14 (22.6%)	 5 (83.3%) 42 (79.2%) 48 (77.4%)	 1.000
Parity, median (IQR)	0.0 (0.0-2.0)	1.0 (0.0-2.0)	0.494
Serum phosphate, median (IQR)	1.9 (1.2-3.4)	1.9 (1.3-3.4)	0.997
Serum alkaline phosphates, median (IQR)	124.5 (102.0-250.0)	144.0 (81.0-196.0)	0.456
Serum calcium	11(42.3%) 15(57.7%)	18(18.9%) 77(81.1%)	0.013 (95% CI 3.1 (1.2-8.0))
Cumulative duration of BF Mean (SD)	25.5 (11.1)	21.4 (10.1)	0.211

Selected Maternal nutritional habits were also assessed for any association with neonatal hypocalcaemia. Milk, fish and cereal intake was assessed. Cereal preparation habits were also assessed. No significant association was found among the tested variables as shown in the table 9 below

Table 9: Maternal nutritional habits association with neonatal hypocalcaemia

Variable	Low	Normal	OR (95% CI)	P value
Milk taken in last 24 hrs				
Yes	18 (69.2%)	77 (81.1%)	0.5 (0.2 - 1.4)	0.746
No	8 (30.8%)	8 (18.9%)		
Fish taken in last 24 hours				
Yes	1 (4.0%)	3 (3.2%)	1.3 (0.1 - 12.8)	1.000
No	24 (96.0%)	92 (96.8%)		
Eggs taken in the last 24 hours				
Yes	4 (15.4%)	12 (12.6%)	1.3 (0.4 - 4.3)	0.713
No	22 (84.6%)	83 (87.4%)		
Beans				
Yes	15 (57.7%)	61 (64.2%)	0.8 (0.3-1.8)	0.542
No	11 (42.3%)	34 (35.8%)		
Green grams				
Yes	1 (3.8%)	3 (3.2%)	1.2 (0.1-12.3)	1.000
No	25 (96.2%)	92 (96.8%)		
Soya				
Yes	0 (0.0%)	2 (2.1%)	-	1.000
No	26 (100.0%)	92 (97.9%)		
Cereals boiled without soaking				
Yes	18 (69.2%)	68 (71.6%)	0.8 (0.3-2.3)	0.815
No	8 (30.8%)	27 (28.4%)		
Cereals soaked				
Yes	7 (29.2%)	23 (24.5%)	1.3 (0.5-3.4)	0.637
No	17 (70.8%)	71 (75.5%)		
Cereals fermenting				
Yes	0 (0.0%)	1 (1.1%)	-	1.000
No	24 (100.0%)	93 (98.9%)		
Cereals sprouting				
Yes	0 (0.0%)	1 (1.1%)	-	1.000
No	24 (100.0%)	92 (98.9%)		

Several neonatal characteristics were also assessed for their association with neonatal hypocalcaemia. Weight and serum alkaline phosphatase levels were found to be low among neonates with hypocalcaemia. The association of this two variables was statistically significant ($p = 0.006$, 95% CI 3.1 and $p = 0.034$ respectively). The neonate's gestational age, Apgar score and serum phosphate levels were not significantly associated with neonatal hypocalcaemia (table 10).

Table 10: Association between neonatal characteristics and neonatal hypocalcaemia

Variable	**Calcium level**		**P value**
	Low (n=26)	**Normal (n=95)**	
Gestation age in weeks, mean (SD)	39.3 (1.0)	39.3 (1.1)	0.941
Average weight, mean (SD)	2.9 (0.2)	3.1 (0.2)	0.006
Apgar score, mean (SD)	10.0 (0.2)	10.0 (0.1)	0.623
Serum phosphate, median (IQR)	1.3 (0.9-2.9)	1.5 (1.0-2.4)	0.421
Serum alkaline phosphates, median (IQR)	83.0 (41.0-118.0)	106.0 (72.0-141.0)	0.034

10.0 DISCUSSION

Hypocalcaemia is a significant neonatal problem in developing countries and is a major cause of morbidity and mortality in the neonatal period. In a study by Amotayo in Nigeria a prevalence of 61.8% was found(6). Studies in Iran and Yemen showed a prevalence of 33.5% (35) and 18% (34) respectively. In Kenya, Nabakwe, in her 1996 unpublished work, studied neonatal hypocalcaemia in premature neonates at Kenyatta National Hospital and Pumwani Maternity hospital in Nairobi. She found that 31% of the premature neonates at KNH and 33% at PMH had hypocalcaemia (38). A high prevalence of hypocalcaemia is reported in neonates of mothers with diabetes mellitus and in neonates with birth asphyxia. The causes of early onset neonatal hypocalcaemia are known to be maternal hypocalcaemia, prematurity and birth asphyxia. Hypocalcemia occurs in as many as 30% of neonates with very low birth weight (<1500 g) and in as many as 89% of neonates whose gestational age at birth is less than 32 weeks (3). These indices portray a neonatal population with a high burden of hypocalcaemia.

This study found an overall prevalence of neonatal and maternal hypocalcaemia to be 21.5% and 24% respectively. These findings compare with the study in Iran by Behjat et al that found prevalence among newly born babies to be 33.5%. Likewise, similar findings were found in studies in Yemen (18%) and in the Nabakwe study (31% & 33%), though this was in premature infants.

In this study, maternal hypocalcaemia was significantly associated with neonatal hypocalcaemia (p 0.013, CI 95% 3.1). This was in keeping with Amotayo's findings in Nigeria (6). In her study, she investigated the prevalence of calcium deficiency among pregnant women and their new born babies in Nigeria. She found the prevalence of neonatal hypocalcaemia to be 61.8%

and that of maternal hypocalcaemia to be 47.2 %.(n =34). This was much higher than what we found in this study. She also found an association between the cumulative duration of breastfeeding with maternal hypocalcaemia in that the longer the breastfeeding period, the lower the maternal serum calcium level. This was not so in this study. Apart from Amotayo in Nigeria, many studies did not look at this relationship.

Maternal dietary intake was assessed in this study. The effects of milk, eggs and cereals were assessed. No significant association was found between this and neonatal hypocalcaemia. In a study in Canada, Cynthia A. etal looked at the effects of restricting milk intake in lactating mothers on select nutrients. She found that milk restriction compromised calcium intake in lactating mothers. This can have a profound effect on the neonate's calcium level (31).

In this study, hypocalcaemia was associated with neonates whose birth weight was lower than those with normal calcium levels. This compares with a finding in Taiwan where Yang CY etal investigated the relationship between the levels of calcium in drinking water and the risk of delivering a child of very low birth weight (VLBW). They found a significant protective effect of calcium intake from drinking water on the risk of delivering a VLBW baby (30). This also compares to the findings in a local study where Wairumbi, in his 2001 unpublished work, investigated the effects of calcium supplementation in pregnancy at KNH. He followed 126 participants from 29 weeks of gestation to delivery. He found that there were significantly more babies weighing 3000grammes and above in the calcium group (at 78.8%) compared to the placebo group at only 66.6% (p value = 0.025) (33).

11.0. EXPECTED APPLICATION OF THE STUDY RESULTS

Results will be made available to KNH. This can be a very good basis for the formulation of a hospital policy on calcium supplementation.

12.0. STUDY LIMITATIONS

1. Vitamin D, parathyroid hormone and magnesium likely to affect both neonatal and maternal serum calcium levels were not assayed in this study because of financial constrains. Alkaline phosphates was assayed to try and determine mothers and babies likely to have Vitamin D deficiency
2. Mothers delivered normally are discharged from KNH within 24 to 48hours of delivery. This limited the observation time to this period where as the serum calcium level in the neonate decreases physiologically to reach its lowest between 24 to 72hours. Some of the assessed neonates might not have reached the lowest level by the time of assessment.

13.0 CONCLUSION

1. The prevalence of neonatal hypocalcaemia in the study population was 26 (21.5%) whereas maternal hypocalcaemia was29 (24%).
2. Maternal hypocalcaemia was a high risk for neonatal hypocalcaemia.
3. Neonatal birth weight was related to neonatal serum calcium level.

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9.0 RECOMMENDATIONS

1. calcium supplementation should be offered during pregnancy and lactation
2. Serum calcium levels should be monitored during pregnancy with a view to supplementing those with deficiency.
3. Neonatal calcium levels should also be monitored with a view to intervening in cases of neonatal hypocalcaemia
4. More studies should be done to investigate the impact of maternal diet on maternal and neonatal calcium levels.

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APPENDIX I

DEFINITION OF TERMS

CASE DEFINITION OF HYPOCALCAEMIA

In both the neonate and the pregnant woman, hypocalcaemia was defined as a total serum calcium level below 2.00mmol/l. This was classified as mild moderate or severe depending on the level as shown in table below. In the case of the neonate, any serum calcium level below 2.00mmol/l with any seizure activity as defined below was also classified as severe hypocalcaemia. For the new mother any serum calcium level below 2.00mmol/l with either positive trousseau's sign or chvostek's sign was classified as severe hypocalcaemia

TABLE 11: GRADES OF HYPOCALCAEMIA

Grade of hypocalcaemia	Mild	Moderate	Severe
Total serum calcium level	2.00 – 1.75 mmol/l	1.74 – 1.55 mmol/l	< 1.55mmol/l or < 2.00mmol/l + at least 1 clinical sign

MEASUREMENTS OF SERUM CALCIUM

In case of low serum albumin, a low total serum calcium assay will not be a true reflection. This was corrected using the following formular:

Ca(c) = Ca (m) + (0.8 x (decrease in albumin concentration below normal in g/dl))

Where Ca(c) is the total calcium corrected and Ca (m) is the total calcium measured. Ionized calcium is relevant for all cell function and the body's homeostasis regulates the ionised calcium concentration. It is, however, the total calcium that is measured and it provides a satisfactory assessment of physiologic calcium. (7).

DEFINITION OF CLINICAL TERMS

TROUSSEAU'S SIGN

Trousseau's sign was elicited by inflation of the blood pressure cuff 20mm Hg above the participant's systolic pressure for three to five minutes. The sign is positive when there is flexion at the wrist and at the metacarpal- phalingeal joints, extension at the interphalingeal joints, and adduction of the thumb.

CHVOSTEK'S SIGN

This was be elicited by tapping the skin over the facial nerve anterior to the external auditory meatus. The sign is positive if this causes ipsilateral contraction of the facial muscles in a participant.

NEONATAL SEIZURE

This is considered when a paroxysmal behavior occurs in a newborn. The seizures are classified in four main groups depending on the clinical presentation as shown in the table below.

Differentiation of seizures from non-convulsive movements is made by observing certain characteristics of the seizure in the newborn. Jitteriness is distinguished clinically from clonic seizures by having no associated ocular movements or autonomic phenomena such as salivation. Jitteriness is also stimulus sensitive and its limb tremor can be suppressed by flexing the limb.

Benign neonatal sleep myoclonus occurs in healthy newborn babies during sleep and is aborted by waking the baby.

TABLE 15: TYPES OF SEIZURES

Type of seizure	Clinical signs
Subtle	Eye deviation Blinking, fixed stare Repetitive mouth & tongue movements Apnea Pedaling, tonic posturing of limbs
Tonic	May be focal or generalized Tonic extension or flexion of limbs
Clonic	May be focal or multifocal Clonic limb movements (synchronous or asynchronous, localized or often with no anatomic order to progression) Consciousness may be preserved
Myoclonic	Focal, Multifocal, or Generalized Lightning-like jerks of extremities (upper>lower)

APGAR SCORE

This is a method of systematically assessing the neonate immediately after birth using clinical features illustrated in the table below;

Table 16: Apgar Evaluation of Newborn Neonates

Score	**0**	**1**	**2**
Sign			
Heart rate	Absent	Below 100	Over 100
Respiratory effort	Absent	Slow ,irregular	Good ,Crying
Muscle tone	Limp	Some flexion of extremities	Active motion
Response to stimuli	No response	Grimace	Cough or Sneeze
Color	Blue / Pale	Body pink ,extremities blue	Completely pink

APPENDIX II

INFORMED CONSENT FORM

I am Dr. Kisiang'ani J.W. From the department of paediatrics and child health, University of Nairobi

I am carrying out a study as part of my postgraduate training in the said department. The title of my study is, "Prevalence And Correlates Of early onset neonatal hypocalcaemia At Kenyatta National Hospital". Calcium is very important for you and your baby. I need information about you to enable me to carry out this study. You will be examined and then a sample of blood (2ml) will be taken from your left arm. Also a sample will be taken from the baby's arm. The results obtained will be used to determine whether you have a deficiency. This will help in managing you and others in this hospital.

Your participation is voluntary and it is not a pre-condition to receiving services in this institution now or in future. The study will be done at no added cost to you.

I, _____ give consent to participate in the study as explained to me by _____

Subject's signature: _____

Date: ___ /____ /_____.

APPENDIX III

QUESTIONNAIRE

PART 1

Date: _____

Age in years:_____

Parity (no. of previous pregnancies): _____

Gestational age in weeks:_____

Level of education : _____

0ccupation:_____

Residence: _____

PART 2

1. Have you ever taken calcium as a supplement?

1. Yes 2. No

2. Have you been taking anticonvulsants in the last three months?

1. Yes 2. No

3. Are you on treatment for diabetes?

1. Yes 2. No

4. Are you on treatment for high blood pressure?

1. Yes 2.No

3. Have you experienced any of the following in the last three months?

a. Perioral paresthesia: 1.yes_____ 2. no_____
b. Twitching: 1. yes_____ 2. no _____
c. Carpopedal spasm: 1. yes ___ 2. no_____
d. Tetany: 1. Yes___ 2. No_____
e. Seizures: 1. Yes____ 2. No_____

4. Do you spend time in the sun? 1. Yes ___ 2. No ____

If so, how much time?

f. Less than 1 hour in a day: 1. yes____ 2. No____
g. More than 1 hour daily: 1. yes. ____ 2.no____
h. Once a week: 1. yes. ____ 2.no____
i. Once a month: 1. yes. ____ 2.no____

5. A DIET RECALL.

Have you taken any of the following in the last 24 hours?

1. Milk: 1.yes____ 2. No_____

2. Fish: 1.yes_____ 2. No_____
3. Eggs: 1. Yes___ 2. No_____

Cereals and legumes

1. Beans: 1.yes____ 2. No_____
2. Green grams: 1. Yes_____ 2.no. _____
3. Soya 1. Yes_____ 2.no _____

Cereal preparation

1. Boiled without soaking: 1. Yes_____2. no. _____
2. soaked before boiling: 1. Yes_____2._ no._____
3. Fermenting: 1.yes_____2. no_____
4. Sprouting: 1. Yes_____2. no _____

6. How many times do you take the following?

a). Milk: 1. Daily, 2. At least twice a week, 3. Once a week, 4. Twice a month 5. Once a month

6. > once in three months

b). Eggs: 1. daily, 2. At least twice a week, 3. once a week, 4. twice a month 5. Once a month

6. > once in three months

BREASTFEEDING HISTORY (time in months)

1st baby: _____

2nd baby: _____

3rd baby: _____

4th baby: _____

5th baby: _____

MOTHER'S PHYSICAL FINDINGS

Trousseau's sign: 1. Positive ____ 2.Negative_____

Chvostek's sign: 1. Positive _____ 2. Negative _____

Blood pressure _____

NEWBORN'S BIRTH HISTORY AND ANTHROPOMETRY

1. What was the mode of delivery? 1. Normal 2.Vacuum extraction 3. Cesarean section
2. Did your baby cry afterbirth? 1. Yes 2. No
3. How long after birth did the baby cry? 1. <5min. 2 After 30min. 3 after 30 min
4. How are you feeding your baby? 1. Breast feeding 2. Infant formular feed. 3.cow's milk

3. Weight: 1. _____2._____ average._____

4. Height: 2. _____2._____ average._____

5. Head circumference1._____ 2. _____. Average._____

NEWBORN'S PHYSICAL FINDINGS

Seizure activity: 1. Yes _____ 2. No_____

Apgar score (recorded) _____

LAB FINDINGS

1. Serum calcium level: a) mother: _____ b) baby: _____

2. Serum phosphate level: a) mother: _____ b) baby: _____

3. Serum alkaline phosphates: a) mother: _____b) baby: _____

4. Serum albumin a) mother: _____ b) baby:_____

APPENDIX IV

REFERRAL LETTER

NAME:

AGE:

I/P NO:

RE: REFERRAL FOR FURTHER MANAGEMENT

I am Dr. Kisiangani J.W. carrying out a study on the prevalence and correlates of early neonatal hypocalcaemia at Kenyatta National hospital. During the study the above named participant was found to be hypocalcaemic and is therefore referred for urgent attention.

Attached please find a copy of the participants' laboratory findings.

Yours sincerely,

Dr. Kisiangani J.W.

APPENDIX V

TABLE 14: BUDGET

Item	**No**	**Unit cost (Kshs)**	**Total (kshs)**
Weighing scale	2	3,000	6,000
Measuring board	2	1,500	3,000
measuring tape	2	500	1,000
Stationary	4 (reams)	500	2,000
Assistants	2	5,000 (@/ mo)	30,000
Laboratory charges			145200
Data management & analysis			20,000
Contingency (15%)			12,300
Total			**207,200**