A DISSERTATION SUBMITTED IN PARTIAL FULFILLMENT OF THE REQUIREMENTS OF MASTER OF PHARMACY IN CLINICAL PHARMACY, DEPARTMENT OF PHARMACEUTICS AND PHARMACY PRACTICE, UNIVERSITY OF NAIROBI.

PREDISPOSING FACTORS OF RICKETS IN INFANTS AND CHILDREN UNDER FIVE YEARS ADMITTED AT KNH:

BY: DR. MWESIGYE JOHN PATRICK
Reg. No. U59/7883/06

DECLARATION

I Certify that this is my original work and has not been submitted anywhere for the same purpose.

Signed -..... Date 27/05/09

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DEDICATION

I dedicate this great work to my beloved wife MBABAZI Hope, Daughter UMWALI. I. Faith and Son BAGABO. I. Kevin, and to you all, as fruits of your sacrifice in one way or another, with all my heart.

ABSTRACT

Background: Rickets is defined as the softening and weakening of bones in children, usually because of an extreme and prolonged vitamin D deficiency. From a few hospital studies including Kenyatta National Hospital (KNH), the estimated prevalence of rickets is 50 – 60% amongst children with prematurity or malnutrition. KNH is the largest referral hospital in East and Central Africa and is located in Nairobi, Kenya.

Objective of the Study: To find out the major predisposing factors to rickets in children at KNH.

Study Design: The study was a cross-sectional study in the paediatrics wards at Kenyatta National Hospital. The study population consisted of infants and children between 3 months and 5 years of age, who were diagnosed and admitted with rickets during the study period. Systematic random sampling was used to obtain a sample of 94 children. The data collected were analyzed using SPSS 12 software.

Results: Predisposing factors of rickets in children and infants in this population were found to be: poverty in families and communities, inadequate nutrition and sunlight exposure, malnutrition, mother's pregnancy and postpartum nutritional factors, birth spacing between siblings, age of 4 months to 15 months, prematurity, early weaning to solid food stuffs and short duration of exclusive breast feeding. Some role was played by previous diseases in the children and their mothers.

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DEFINITION OF TERMS

i-	Infant	A baby below one year of age
ii-	Children	Boys and girls below age of puberty
iii-	Health Professionals	Medical Doctors, Pharmacists, Nurses, and Other Medical Paramedics
iv-	Care taker	Any one looking after the baby
v-	Preterm	Birth of a baby before 37 weeks of gestation
vi-	Osteomalacia	Softening of the bones due to lack vitamin D
vii-	Kyphoscoliosis	Abnormal curvature of the spines both forwards and sideways
viii-	Malnutrition	A condition resulting from lack of food nutrients for proper body growth and development
ix	Hidrosis	Excessive sweating
x	Flaccidity	Lack of firmness
xi	Marfan syndrome	Inherited abnormality of connective tissue
Xii	Harrison's sulcus	a depression on both sides of the chest wall of a child between the pectoral muscles and lower margin of the ribcage.

Abbreviations and acronyms

UV Rays	Ultraviolet Rays
KNH	Kenyatta National Hospital
VDR	Vitamin D Receptor
G	grams
VDD	Vitamin D Deficiency
VDI	Vitamin D Insufficiency
EBM	Exclusive Breast Milk
mo	months
ASF	Animal Source Foods
CRSP	Collaborative Research Support Program
UK	United Kingdom
RDA	Recommended Daily Allowance
DNA	Deoxyribonucleic Acid
SCI	Spinal Cord Injury
MS	Multiple Sclerosis
NLSY	National Longitudinal Survey of Youth
IHDP	Infant Health and Development Program
PEM	Protein Energy Malnutrition
PEW	Paediatric Endocrinology Ward

CHAPTER 1

1.0 Introduction:

Rickets is defined as the failure of osteoid to calcify in a growing person; it is also seen as the softening and weakening of bones in children, usually because of an extreme and prolonged vitamin D deficiency.[1,2]

Rickets occurs when the metabolites of vitamin D are deficient. Less commonly, dietary deficiency of calcium or phosphorus may produce rickets. Vitamin D (cholecalciferol, vitamin D-3) a steroid compound is formed in the skin under the stimulus of ultraviolet light.[2]

Vitamin D is essential in promoting absorption of calcium and phosphorus from the gastrointestinal tract, which children need to build strong bones. A deficiency of vitamin D makes it difficult to maintain proper calcium and phosphorus levels of the bones.[1]

When the body via the hypothalamus senses an imbalance of calcium and phosphorus in the bloodstream, it reacts by taking calcium and phosphorus from the bones to raise blood levels to where they need to be. This softens or weakens the bone structure, resulting most commonly in skeletal deformities such as bowlegs or improper curvature of the spine.[1]

If vitamin D or calcium deficiency causes rickets, adding vitamin D or calcium to the diet generally corrects any resulting bone problems for the child. Rickets due to a genetic condition may require additional medications or specialized treatment. Some skeletal deformities caused by rickets may need corrective surgery.[1,3] Ultraviolet light was the only significant source of vitamin D until early in the 20th century when ergosterol (vitamin D), was discovered.[2] Rickets of prematurity and maturity terms in children is currently well recognized worldwide. [4,5] This has not come by accident, but it is due to improved neonatal care, which has seen and enabled more low birth weight infants to survive through the neonatal period.

The severity of the disease ranges from mild under mineralization (known as osteopenia), to severe bone disease with fractures.[6,7]

The incidence of rickets varies from one centre, country, region and continent to another, as a result of differences in the levels of advancements in health facilities and health care services offered in different areas of the world. It appears also to vary according to the

socioeconomic and, sociodemographic settings (locations) and the birth weights of the infants.[5]

However, the etiology of rickets of prematurity is controversial and not precise. Rickets of prematurity has been reported in premature infants fed on breast milk and artificial infant formulas with or without vitamin D supplementation[8,9,] Inadequate intake or malabsorption of phosphate.[10,11] calcium[12,10] or vitamin D, has variously been implicated in the pathogenesis of rickets of prematurity and rickets in general.[1]

Renal function in both infants and children tends to be poor and vitamin D may not promote tubular reabsorption of phosphate. In addition, because of the small size, low birth weight babies, many of whom are preterm, and other children in some communities, are less exposed to sunlight, making their diet the sole source of vitamin D which may result in complicated rickets.[6,13] Their diet of breast milk or infant formula without any other supplements of vitamin D and minerals is an inadequate source of these essential compounds.[8,9]

1.1 History

Rickets was known as far back as early antiquity. Soranus of Ephesus (early 2 century A.D) described leg and spinal deformities in children. Galen (130 – 201 A.D) described rachitic changes in the oseous system including thoracic deformities in a greater detail.[6, 2]

In the 15th century the children of England were widely found affected by rickets and descriptions of severe forms of the disease began to appear with increasing frequencies in the work of English physicians and this fact warranted calling rickets the "English Disease" [6,14] The first work on the clinical aspects and anatomy of rickets belongs to an English physician Francis Glisson (1650).

Other great contributions in this disease condition have been by the Russian physicians. In his paediatrics publication in St. Petersburg in 1847, S.F. Khotovitsky not only described in detail the skeletal changes, but also indicated such concomitant symptoms of rickets as hidrosis, muscular flaccidity, retarded motor development, changes in gastrointestinal tract and many others.[6]

Rickets appeared in epidemic form in temperate zones when the factories of the Industrial Revolution produced so much smoke that ultraviolet rays were blocked. Rickets probably became the first childhood disease caused by environmental pollution. At that point in history, the deficiency of sunlight could be said to have caused the rickets epidemic in industrial areas.[2,6] Although rickets was formerly believed to be a disease of the moderate zone of Europe and America and other highly humid areas, this view was found to be incorrect. Rickets occurs in countries located in the middle latitudes, in the cold and the south too.[6,14]

About a century ago, N.F Filatov attached enormous importance to rickets as one of the causes of high child mortality. In his letter to S.P Bolkin in 1888 he wrote "I am inclined to believe that children who die in their second year of life and later almost all have or had rickets and it is therefore no less important to eliminate the causes of rickets than it is to control diarrhoea and other high morbidity and mortality diseases in children".[6]

1.2 Manifestations

Vitamin D is the major steroid hormone that controls calcium homeostasis, and plays a crucial role in normal bone growth, calcium metabolism, and tissue differentiation; it exerts its actions through the vitamin D receptor (VDR).[2]

Its deficiency results in failure of mineralization of the growing bone or osteoid tissue. Subsequently, excessive but calcium deficient, osteoid tissue is laid down resulting in softening of the bones with deformities, fractures, and bone tenderness.[2,6] The early characteristic changes are seen radiologically at the metaphgses of the long bones, and as a result of demineralization of the shafts.[1, 6]

Clinically, the earliest bone changes include: craniotabes and enlargement of the epiphyseal cartilages of the long bones especially the ribs, the fibula and tibia, the radius, ulna and prominent wrist.[6,15] If rickets progresses at a later stage, thickening of the skull develops.[15] This produces frontal bossing, and the closing of the anterior fontanelle is delayed. The laying down of uncalcified osteoid at the metaphysis leads to spreading of those areas, producing knobby deformity.[2,16]

In the chest, knobby deformity results in the rachitic rosary. The weakened ribs pulled by muscles also produce flaring over the diaphragm, which is known as Harrison groove. The sternum may be pulled into a pigeon-breast deformity. In more severe instances in children older than 2 years, vertebral softening leads to kyphoscoliosis.

The ends of the long bones demonstrate some knobby thickening resulting in Marfan's syndrome, a serious heritable disorder of connective tissue with manifestations in many organs including the eyes, heart, aorta, skeleton, skin, and lung. Because the softened long bones may bend, they may fracture at one side of the cortex (ie, greenstick fracture).[2,14]

The following also may be observed: Excessive sweating, irritability, widened fontanelles, pot belly due to hypotonia of the abdominal muscles and bowing of the legs when they start to support truncal weight on the lower soft supporting bones.[17,18]

Delay in achieving physical developmental milestones does occur in late stages of the child's life.[11,13]

1.3 Diagnosis

In some countries, like China, a clinical standard for diagnosing rickets based on a list of signs and symptoms has been developed. Primary signs – leg pain on walking, frontal bossing, pectus carinatum, funnel chest, rachitic rosary, Harrison's sulcus, bowed legs, X-shaped legs, and widened wrists;
Secondary signs – occipital rubbing, anterior fontanelle closing after 18 months of age, teeth eruption after 10 months of age.[1,6]

1.3.1 Differential Diagnosis:

- Osteoporosis
- Metastatic bone disease
- Primary bone malignancies (lymphoma, myeloma).[19]

Various modalities have been used to make early and precise diagnosis of rickets in all age groups, infants and children included.

These include chemical assessments, biochemical blood assays of calcium, phosphorus and alkaline phosphatase, radiological assessments of long bones and electrochemical assays of the long bone's mineral contents.[1,11]

1.3.2 Laboratory:

- Alkaline phosphatase - increased
- Serum calcium is low or normal (never high)
- Hypophosphatemia
- Aminoaciduria
- Acidosis
- Glucosuria
- Hypouricemia.[19,20]

Craniotabes, one of the clinical signs of rickets is reported to occur in 6-8 weeks of life, in some patients with rickets.[2,15] Clinical features, however, become obvious in the late stage of the disease, and in early stages only alkaline phosphatase has been found to be useful in the diagnosis of rickets. It has therefore received a lot of attention to improve on its

diagnostic value/ roles too and more routinely assayed in evaluation of patients suspected of having rickets.

Serum alkaline phosphatase, especially the bone isoenzyme, is generally high in children due to increased bone turnover secondary to rapid growth process.[19,21] It may be less reliable in the diagnosis of rickets in preterm, if taken on its own, especially if the cut off value used for older children is employed.

Alkaline phosphatase levels have been found normal in some patients with radiological evidence of rickets and evaluated in others where rickets has not been demonstrated by other methods. It may be normal in infants with rickets if serum proteins levels are low. However, it is presumed that in some of these reports, the inability to confirm the presence of rickets despite high levels of alkaline phosphatase, could have been due to the use of radiology in particular, X- ray for this purpose.[6,22]

Even though radiological assessment of the long bones is diagnostic quite early and reliable in evaluation of rickets, it has not been the most sensitive method and misses out some patients with mild metabolic diseases.[14]

Bone biochemistry and photon absorptiometry are currently the best diagnostic procedures for rickets of both prematurity and childhood but their use is limited due to their availability in few centres and the fact that they are too expensive for routine use.[13,21]

1.4 Prevalence

A recent survey showed that 20–34% of South East Asian children had biochemical evidence of vitamin D deficiency. However, there is no information, other than case series, on the prevalence of clinical rickets among non-caucasian children in the UK, and is reported to be of sporadic nature in many developed western countries.[3,14]

In Kenya, studies carried out in various hospitals in 2003 estimated that about 58.8% of premature children and infants have rickets, Neighbouring Ethiopia has rickets about 41% of paediatric out patients and the highest prevalence of 87% has been reported in Inner Mongolia.[3,23]

1.5 Causes and predisposing factors in rickets

1.5.1 Overall Causes:

Rickets can be caused by a wide variety of pathogenic processes including, but not limited to, vitamin D deficiency (reduced exposure to sunlight, poor nutrition, malabsorption syndromes)

Defective metabolism of parent vitamin D to active metabolites (drug-induced, e.g., anticonvulsants - phenytoin, or chronic renal failure), hypophosphatemia (renal tubular acidosis, hypophosphatemic syndrome) and miscellaneous causes (long-term hemodialysis, malnutrition, vitamin D-dependent rickets) are also well established causes.[20,24]

1.6 Predisposing Factors:

These include for example: Prematurity, poverty in families and communities, Inadequate nutrition and sunlight exposure,[6] chronic renal disease,[20] epilepsy,[24] malnutrition,[10] previous gastric surgery, chronic diarrhoea for more than 14 days, pregnancy-nutritional factors, etc.[25,26]

1.7 In developed countries

Rickets is found in dark-skinned and unsupplemented individuals in the north (African – Americans) and among unsupplemented, poorly nourished, vitamin D deficient individuals [24,25]

1.8 Known causes of rickets in Kenyan Children.

Multiple factors are at play here, including a low intake of milk and hence of calcium and phosphorus, premature birth, dark skinned and perhaps reduced exposure to sunshine and ultraviolet light, vitamin D deficiency, nutrition complications, and some anticonvulsive drugs involvement. All these factors operate jointly, but the lack of milk in the diet was a major factor.[22,27]

1.9 Prevention / Avoidance:

Although most adolescents and adults receive much of their necessary vitamin D from exposure to sunlight, infants and young children need to avoid direct sun entirely or be especially careful by always wearing sunscreen to avoid development of skin cancer. Ultraviolet B light triggers a modification of a cholesterol-related molecule located in the

membrane of skin cells. The vitamin D that is created is transformed into 25-hydroxyvitamin D, in the liver.

The kidney, as well as some other tissues, further converts this precursor into 1,25-hydroxyvitamin D, the most physiological active vitamin-D metabolite, which is also called calcitrol.[28]

1.10 Vitamin D supplements

Since human milk contains only a small amount of vitamin D, the American Academy of Paediatrics recommends that all breast-fed infants receive 200 international units (IU) of oral vitamin D daily beginning during the first two months of life and continuing until the daily consumption of vitamin D-fortified formula or milk is two to three glasses or 500 milliliters (ml).

Vitamin D supplements for infants generally come in droplet form. Only supplements that contain up to 400 IU of vitamin D per ml or tablet should be used, supplements containing a higher concentration of vitamin D (some forms come in levels of up to 8,000 IU/ml), should be avoided because they are unsafe for children.[28]

1.11 Getting enough calcium

Calcium and phosphorus consumption are also important for bone formation in childhood. Breast milk is the best source of calcium during a child's first year of life. Most commercially available formulas also meet calcium requirements. Because of these factors, infants in the United States generally achieve 100 percent of their recommended intake of calcium.

Recommended daily intake of calcium is as follows (serving sizes vary with age):

- 1 to 3 years of age. 500 milligrams (mg) (two servings of dairy products a day)
- 4 to 8 years of age. 800 mg (two to three servings of dairy products a day)
- 9 to 18 years of age. 1,300 mg (four servings of dairy products a day, as most bone mass production occurs during this period)
- 19 to 50 years of age. 1,000 mg a day (three servings of dairy products a day)

Milk accounts for three-fourths of the calcium in the food supply of the United States. Other sources of calcium include leafy green vegetables (spinach), fortified orange juices, fortified breakfast cereals and calcium supplements.[4,26]

1.12 Possible Complications:

While easily treated once it is diagnosed, rickets has a severe list of complications if left untreated. Untreated vitamin D deficiency rickets may lead to:

- Increased susceptibility to serious infections [20]
- Delays in child's motor skills development [6]
- Failure to grow and develop normally [6]
- Skeletal deformities
- Chronic growth problems that can result in short stature (adults measuring less than 5 feet tall)
- Seizures
- Dental defects.[1, 26]

The high prevalence of chronic vitamin D deficiency in north China potentially has been found to put many children at increased risk, of diabetes, hypertension, and a host of cancers.[1]

1.13 Study justification:

Despite the advances in health facilities and economic achievements observed in Kenya, rickets still remains a health hazard to infants and children's life in various families in different parts of the country. Knowledge of the principal pre-disposing factors in this area would enable them to be tackled and the disease burden reduced. There is general knowledge about these factors from a few studies done, but there is need to have an update and more specific study in this hospital.

Therefore this study aimed at finding out the extent and the nature of the predisposing factors to rickets in children, so that appropriate measures can be taken to prevent and manage the problem.

The results from this study are expected to have a positive contribution in caring and providing to rickets patients through:

i- Development of guideline manuals regarding the underlying factors in rickets, for both health professionals, parents of the children and the general public.

ii- Helping in the identification of the real local risk factors that predispose children to rickets in the communities.

2.0 Literature review:

In Kenya, studies on rickets have been carried out in early 1900s, 1930s, 1950s and the 1980s with the latest in 2000 – 2003. In this last study called the Child Survival Project, of the 324 children registered, 24 were clinically diagnosed with rickets and these included three sets of twins. In KNH, 604 and 702 infants and children under five years were admitted with rickets in 2004 and 2005 respectively.[29]

Like in any other developing countries, rickets in infants and children under five years in Kenya, has been attributed to various predisposing factors such as, unfavourable living conditions, dark and over crowded housing, non or little exposure to uv light, starvation (inadequate diet with insufficient vitamin A, D, C and B is particularly important. More so is the mother's health status during pregnancy and breast feeding period has been indicated too), chronic diseases, including malnutrition, short breastfeeding duration, vegetable and cereal-based complementary foods with negligible cow's or goat's milk, and confinement indoors when the mothers were cultivating the fields.[22,27] Very dark skinned or heavily clothed and completely covered infants and children, have been indicated to have high chances of developing rickets compared to their counterparts, rickets develops sooner and runs a very severe course in these infants and children.[4,6,]

Prolonged use of some anticonvulsants and aluminium containing antacids, and any other drugs and substances that would cause a chronic increase in gastrointestinal bowel movements, for example laxatives, causes a major rise in the incidence of second and third degree rickets.[24]

Reports of hypovitaminosis D associated with anticonvulsant drugs in paediatric patients were found conflicting in some studies. The effects of carbamazepine or sodium valproate on vitamin D status were evaluated prospectively in 51 ambulatory epileptic children who were followed during the first year of the study and in 25 and 6 children during the second and third year, respectively. Serum 25-hydroxyvitamin D, parathyroid hormone, calcium, and phosphorus levels were determined before and every 3 months during anticonvulsant therapy. Subjects were grouped into four classes (0, 1, 2, and 3 consisted of the patients before and during the first, second, and third years of the treatment, respectively). The control group consisted of 80 healthy children. Comparisons

between controls and patients of class 0 for the means for each season of all variables showed no significant differences. A decreasing trend in serum 25-hydroxyvitamin D ($P < .03$) and an increasing trend in serum parathyroid hormone ($P < .04$) levels were noticed in all seasons from class 0 to class 3. Twenty-five patients (49%) acquired hypovitaminosis D during the study period. The effects of seasonality on serum 25-hydroxyvitamin D, parathyroid hormone, and calcium were noticed in our patients grouped in classes 0, 2 and 3, as well as in controls. The study showed that carbamazepine or sodium valproate can cause hypovitaminosis D in children.[24]

Rickets has been found in severest form in infants of birth weight of 1000g and less, in whom radiological features of rickets and frank fractures have been reported in up to 57% of cases.[22] In one retrospective study, 32% of the surviving low birth weight infants (where birth weight was less than or equal to 2000g), had biochemical and radiological changes, suggestive of rickets.[6]

In his study on the prevalence of rickets at Kenyatta National Hospital, Dr. Donald P.O. Oyatsi investigated patients between the ages of three months to nine years, admitted at paediatric endocrinology ward (P.E.W), with rickets. He recruited twenty nine patients into the study, out of whom 58.6% were aged two years and below. The etiological factors of rickets in these patients were found to be interrelated.[5] Phillip reported 45 cases of rickets through the analysis of records in one hospital in Central Province in 1926–1930.[30]

The Nutrition Collaborative Research Support Program CRSP in Kenya project did not find clinical evidence of rickets in the Embu children that were studied in the early 1980s. It was surprising that several cases of rickets were encountered in the recent Child Survival Project (CSP), carried out in the same area in Embu.[30] The toddlers were enrolled in a feeding intervention study from 2000–2003. Twenty-four such cases were diagnosed clinically, out of 324 children registered for the CSP project. The children with rickets shared the following risk factors: short breastfeeding duration, vegetable and cereal-based complementary foods with negligible cow's or goat's milk, and confinement indoors when the mothers were cultivating the fields. The children, aged 18–36 months, included three sets of twins.

Multiple factors are at play here, included a low intake of milk and hence of calcium and phosphorus, [30,31] no intake of ocean fish hence a low vitamin D intake, [27] low birth weight, [8]

and perhaps reduced exposure to sunshine and ultraviolet light. [6] All these factors operate jointly, but the lack of milk in the diet was a major factor.[2,30] The affected children were provided with milk supplements and vitamin D-3 for 1 month, which caused a noticeable regression of their rickets.[31]

As part of addressing the problem of low milk intake in primary school and kindergarten children, the daily provision of milk to children during midmorning sessions at school was a promising strategy, in alleviating the poor nutrition.

The program was started in 1982 by the president of the Republic of Kenya, to alleviate hunger among children, many of whom went to school on an empty stomach and appeared to have difficulty concentrating in class in the late morning. This program continued for several years in the 1980s but was then abandoned for lack of funds to sustain it. At present, only sporadic and short-term school milk programmes are in existence, usually sponsored at times of drought by the World Food Program.[27]

The Nutrition Collaborative Research Support Program (CRSP) longitudinal observational study in Embu entitled, "Energy Intake and Human Function," (1982–1984), focused on mild-to-moderate energy malnutrition. Quantitative data were collected on food prepared and consumed by household members, Toddlers, and to a limited extent school going children, were followed whenever possible to observe and record even self-obtained snacks.

The diet in the Embu CRSP households was generally low in energy and fat (animal and vegetable), and extremely low in Animal Source Foods (ASF). Cow's milk and goat's milk were the main sources of animal protein, consumed mainly in sweet tea, and in small quantities.[27,32]

Maize was the main staple, complemented by beans. Millet and sorghum, used elsewhere in Kenya, were much less frequently consumed than maize. The phytate and fiber content of the diet was extremely high and this sharply impairs calcium absorption. Potatoes and various vegetables were eaten when available. Fruits such as mango, papaya, avocado and banana were consumed in season.[12,32]

Energy intake was particularly low in most of the households. Boys and girls aged 8–9 yrs had energy intakes that ranged around 78% of their Recommended Daily Allowance (RDA),

with only 6% of the energy coming from Animal Source Food (ASF). The situation was worse among the toddlers aged 18–30 months, whose energy intake met 73% and 70% of their RDA, for boys and girls, respectively.[27]

ASF, meat, fish and fowl were rarely if ever consumed. Hence there was an inadequate intake of micronutrients: iron, zinc, vitamin A, vitamin B-12, riboflavin and calcium. The dietary iron and zinc was made relatively unavailable by the high phytate and fibber in the diet. Anaemia was common, due to iron deficiency, but compounded by malaria and hookworm infestation. Mean haemoglobin levels were 121, 107 and 109 g/L for the same childhood age groups, respectively, with a prevalence of anaemia over 50% in all groups.[27, 32]

Toddlers' aged 12–30 mo demonstrated very little severe malnutrition, but 27% and 30% were stunted and moderately underweight, respectively. Stunting started early during infancy. Zinc deficiency, which was not known to exist in Kenya before this study, may have played a role in the stunting among these children. There was no clinical evidence of vitamin A deficiency or rickets. However, malnutrition was encountered depending on the season with 27–48% of all school-age children moderately malnourished and 1.5% severely so.[32]

Conversely, children who consumed ASF, particularly meat, performed better on cognitive tests, had better growth and less morbidity, were more attentive in the classroom and more active in the playground than those who consumed no ASF. This association remained positive even after statistical control for a large number of covariables and confounding factors such as socioeconomic status, morbidity and parental literacy. Stunting was prevalent, occurring in 30% of the children. These findings suggest the benefit of ASF in the diet of the Kenyan children.[27,32]

Nonetheless, the above findings were based on observational studies. A randomized controlled feeding intervention study was needed to establish a causal relationship between ASF, both meat and milk, and functional outcomes. This led to the randomized controlled intervention feeding study to test whether there was a causal relationship between intake of ASF, such as milk or meat, and cognitive function, activity, growth, development and morbidity, because a literature review showed persistent lack of ASF in the diets of young and older children, and the predominance of carbohydrate in the cereal-based diets.[32]

Breast feeding was often found interrupted early by the introduction of porridge or gruel from cereals, with little or no addition of cow's or goat's milk. As the child grew, the main foods remained cereal-based, initially as over diluted porridge/gruel and later, as bulky solids. The diluted foods had low energy content, whereas the bulky solid foods could not be eaten in large enough amounts to provide the required energy. Feeding frequency may not have been frequent enough to provide adequate quantities of nutrients. The solid foods were not eaten with milk. Almost no meat, fish or fowl was consumed by children even if available.[27,32]

It has been found, that in breastfed children the assimilation of calcium and phosphorus salts is higher, being at about 70% and 50% respectively, while it is about 30% and 20 – 30% respectively in those on artificial feeding.[6,31]

In a number of studies, results have shown that severe vitamin D deficiency (VDD) causes rickets in infants and children, due to decreased bone mineralization. VDD in pregnancy is associated with restricted foetal and infant growth, and predisposes to neonatal VDD and hypocalcaemia. Vitamin D status in childhood and adolescence may play a role in the prevention of osteoporosis. Adequate status may reduce the adult risk of diabetes, ischemic heart disease, hypertension and tuberculosis.[1,30]

In Melbourne, nutritional rickets was documented during the 1960s; 70% of the affected children were migrants of Mediterranean origin. More recently, VDD has been documented in veiled or dark skinned pregnant women, and in immigrant infants from different backgrounds presenting with rickets.[30]

In the absence of supplementation, skin pigmentation and exposure to solar ultraviolet B (UVB) irradiation determine serum levels of 25-hydroxyvitamin D (25-OHD) through endogenous production.[33]

Throughout history, many healing modalities have cycled in and out of favour. Sunlight (heliotherapy) was often a part of mankind's healing armamentaria, including that of Hippocrates, the Father of Western Medicine. In the early twentieth century, heliotherapy was used to treat many disorders, such as tuberculosis, rickets in children, and war wounds. Its importance was underscored when Dr. Niels Finsen was awarded the 1903 Nobel Prize for developing an ultraviolet (UV) treatment for tuberculosis.

Boston's Floating Hospital got its name because it originally was located on a floating boat, in which children with rickets could sunbathe. At that time, many hospitals were built to allow access to more sunlight. Sunlight remains an important hospital-design consideration.

However, as our increasingly pharmaceutically oriented medical profession developed powerful, infection-fighting antibiotics and drugs, and as milk and other foods were supplemented with vitamin-D, heliotherapy faded to the background. Its decline was greatly accelerated by the fear of acquiring skin cancer.[34]

Ultraviolet Light

Sunlight is composed of electromagnetic radiation of varying wavelengths, ranging from the long-wavelength infrared light to the short-wavelength ultraviolet. The ultraviolet light is further subdivided into UVA and the even shorter-wavelength UVB radiation. Although UVB causes sunburns, it is also the component that initiates Vitamin-D production in the skin.

Exposure to vitamin-D-producing UVB light can vary greatly depending upon many factors, including time of day and year; and the latitude, altitude, and prevailing weather conditions of where we live.

Latitude is especially important. For example, persons living north of about 37° (roughly, a line from Richmond to San Francisco in USA), will be exposed to little UVB from at least November through February because the sun's zenith angle is so low that the atmosphere absorbs most UVB.

Because it is fat soluble, excess, sun-produced, vitamin D is stored in body fat, and, to some degree, can be later used in sun-deficient periods.

Vitamin-D Production and Metabolism

The process by which vitamin D is produced and exerts its biological effects is complicated, involving several vitamin-D-related molecules.

Ultraviolet B light triggers a modification of a cholesterol-related molecule located in the membrane of skin cells. The vitamin D that is created is transformed into 25-hydroxyvitamin D, in the liver.

The kidney, as well as some other tissues, further converts this precursor into 1,25-hydroxyvitamin D, the most physiological active vitamin-D metabolite, which is also called calcitrol. Traditionally, calcitrol is understood as a hormone that, together with parathyroid hormone, regulates blood calcium levels and, in turn, bone density. In this role, calcitrol targets the intestine, where it promotes calcium absorption; and bone, where it catalyzes calcium release to help restore depleted blood calcium levels.

However, recent studies indicate that viewing calcitrol just in this role is quite limited. In fact, the hormone exerts a plethora of biological effects on diverse tissues, implying that this sunlight-generated hormone sustains health throughout the body.

Although much remains to be understood, circulating calcitrol enters cells and complexes with the genes in the cell nucleus. This affects DNA expression and, in turn, overall cell functioning and growth. Because calcitrol maintains normal cell proliferation, it inhibits cancerous growth. In addition, calcitrol influences immune-cell activity, helping to explain vitamin D's seemingly beneficial role in infectious disease and immune-related disorders, such as multiple sclerosis, rheumatoid arthritis, and diabetes.

Given these considerations, the implications of preventing vitamin-D deficiency, either through sunlight or diet, are now more profound than ever.

Vitamin-D Sources

Relatively few foods naturally contain vitamin D, the most abundant being oily fish like salmon, sardines, and mackerel. Because of limited natural sources, numerous foods, such as milk, are vitamin-D fortified.

Many scientists now believe we need 1,000 IU per day of vitamin D (IU = international units, to avoid deficiency. For reference, a salmon serving contains about 360 IU, a glass of fortified milk about 100 IU, an egg 25 IU, and a tablespoon of cod liver oil 1300+ IU.

In comparison, full-body sunbathing for a period of time that will just make a white person turn pink will produce 10,000-20,000 IU of vitamin D, equivalent to 100 to 200 glasses of fortified milk. Given such copious production, relatively casual sun exposure (arms, etc) should meet vitamin-D needs.

However, no vitamin D will be generated in short-daylight months above certain latitudes. In these periods to avoid vitamin-D deficiency, it may be necessary to consume supplements or use artificial farming.

The importance of solar-produced vitamin D was underscored in a study that evaluated vitamin-D status in a submarine crew after two months of acute sun deprivation. Although the crew consumed a vitamin-D fortified diet, their levels of this nutrient plummeted.

Vitamin D can be toxic if too much is consumed from supplements or mistakenly over-fortified foods. However, prolonged sun exposure does not generate toxic vitamin-D levels

At-Risk Groups

Although everyone is vulnerable to vitamin-D deficiency, especially in winter, certain groups are particularly predisposed. First, when exposed to the same amount of sunlight, elderly individuals produce only 20% of the vitamin-D young adults do. As a result more than half of individuals older than 65 are vitamin-D deficient.

Second, due to their skin pigmentation, African Americans require much more sun to produce the same levels of vitamin D than do fair-skinned Caucasians. At least 50% of African Americans, who are also less likely to drink fortified milk due to lactose intolerance, are vitamin-D deficient sometime during the year. Apparently, as a consequence, they have a much greater incidence of disorders associated with reduced sun exposure of northern latitudes.[34,35]

But above all, to have the knowledge of the risk factors in specific populations is important in preventing VDD in pregnant women and infants, and may also contribute to the prevention of osteoporosis and rickets.[32]

The increased rates of VDD in adult East African immigrants living in Melbourne, Australia suggested that their immigrant offspring are also at risk of VDD. Malnutrition, iron and vitamin A deficiency are prevalent in African children, and VDD is associated with underweight and with iron deficiency anaemia.[30]

Consecutive immigrant children aged 0–17 years attending an immigrant health clinic at the Royal Children's Hospital (RCH) were enrolled prospectively. The clinic was established in

consultation with community representatives after a number of health issues including VDD were studied in a community based project involving adult members of the East African community in Melbourne. Potential participants were referred to the clinic by East African community health workers. All enrolled children were indigenous to countries in East Africa (Somalia, Sudan, Ethiopia, Kenya, Egypt, Eritrea or Djibouti). Subjects were enrolled between December 2000 and November 2002.[30]

Information relating to demographics, country of origin and residence prior to migration (including refugee camps), feeding practices, immunization status, medical history, sun exposure, carer's education, carer's concerns about the child's health, and utilization of health services in Australia was obtained.

Results obtained showed: Low 25-hydroxyvitamin D (25-OHD) levels (<50 nmol/l) occurred in 87% of children, and VDD (25-OHD, <25 nmol/l) in 44%. Risk factors included age, <5 years, female gender, increased time in Australia, decreased daylight exposure and winter / spring season. Anaemia (20%), vitamin A deficiency (20%) and iron deficiency (19%) were also identified and eventually all this would lead to rickets among other diseases.[30]
A study by Salimpour showed an incidence of 15 per cent in children less than 5-years-old in Tehran – Iran.[28]

443 patients aged 3–24 months were admitted to the paediatric ward at Queen Alia Hospital Amman Jordan and were included in the study. Male to female ratio was 1.2:1. Forty-seven infants were diagnosed with nutritional rickets, accounting for 10.6 per cent of all children studied.[35]

In a nationwide survey in China from 1977 to 1983, the incidence of nutritional rickets was reported to be as high as 40.7%. In a similar study on children admitted to hospital in South Africa, Sochett, et al. found radiological evidence of rickets in only two of 114 children (1.7 per cent).[31]

Vitamin D deficiency rickets has been reported to be 'China's number two most common nutritional disease of children'. The most conservative prevalence rate of rickets among children under age 5 years in China is reported as 15.9%, with rates among infants of 26.7%,[1] this is a decline from 40.7% in China in 1983. This makes the prevalence of rickets in China one of the highest in the world.[36]

In Britain there were more frequent reports of rickets among children of the low socioeconomic immigrant families/population than in the rest of the population with high standards of living.[6]
Indeed poverty in families and communities with low socio economic status and its role in rickets should not be a surprise, according to some previous studies.

Income poverty is the condition of not having enough income to meet basic needs for food, clothing, and shelter. Because children are dependent on others, they enter or avoid poverty by virtue of their family's economic circumstances. Children cannot alter family conditions by themselves, at least until they approach adulthood.[37] Poor families are more likely to be headed by a parent who is single, has low educational attainment, is unemployed, has low earning potential, and is young.[37]

Studies show poor children suffer higher incidences of adverse health, developmental, and other outcomes than non-poor children. Compared with non-poor children, poor children in the United States experience diminished physical health as measured by a number of indicators of health status and outcomes. In the 1988 National Health Interview Survey, parents reported that poor children were only two thirds as likely to be in excellent health and almost twice as likely to be in fair or poor health as non-poor children.[37]

Although overt malnutrition and starvation are rare among poor children in the United States, deficits in children's nutritional status are associated with poverty. As described fully in the Child Indicators, stunting growth (low height for age), a measure of nutritional status, is more prevalent among poor than non-poor children. Studies using data from the NLSY show that differentials in height for age between poor and non-poor children are greater when long-term rather than single year measures of poverty are used in models to predict stunting. These differentials by poverty status are large even in models that statistically control for many other family and child characteristics associated with poverty.[38]

As reported in one study, children living below the poverty threshold are 1.3 times as likely as non-poor children to experience learning disabilities and developmental delays.[39] Reliable measures of cognitive ability and school achievement for young children in the Children of the National Longitudinal Survey of Youth (NLSY) and Infant Health and

Development Program (IHDP) data sets have been used in a number of studies to examine the relationship between cognitive ability and poverty in detail.[27,39] A study carried out in Bangladesh tried to find out the food habit of each household both in tribal and plain land households. It was found that rickets was more common in low-socioeconomic group where dry fish was commonly used. Dry fish is preserved with some chemicals. So there might have been some correlation with those two. Out of 35 tribal households they did not get any rickets children, on the other hand and of 35 plain land houses hold, got in the sample households 17% of rickets children and referral 11% of rickets children. Major difference in the food among the tribal and plain land household, was that tribal people took maximum vegetables than the Plain land household (47%). Dry fish was also less among the tribal people (26%). The tribal people in comparison to the plain land household took less chillies and spices (36%). And their cooking time was less than the plain land people. Mostly tribal people eat most of the wild animal and all types of sea fish, snail, snake, shark etc 100%. Another observation was that tribal people used less dress than the plain land people; it meant more exposure to sunlight. Birth spacing in tribal was more, as result children got maximum time to have the breast milk. It was also observed that tribal People spent only 6% in education, 68% to the food and to the health17% sector from their income. On the other hand Plain land people spent 57% for the food, 16% for education and for health 17%.[3]

In the international public health and development communities, rickets has become something of a forgotten disease - it tends to be thought of in historic terms without the recognition that in many places it continues to be a source of disability today. This may be due to the virtual elimination of the disease from industrialized countries, which has been achieved through the development of infant care and food systems that provide growing children with adequate amounts of the essential nutrients they require to grow healthy bones (calcium, phosphorus, magnesium, and vitamin D). However, to forget about the disease in the wake of those successes would indeed be short-sighted, as it is clear that rickets persists as a public health problem in less developed countries.[37]

Unlike other nutritional diseases, rickets can be cured but not healed. That is, the underlying metabolic lesions can be corrected (by using diets containing adequate amounts of calcium and/or vitamin D); however, without orthopaedic treatment, such children are often left physically disabled for life. In this manner, the disease casts a long

shadow, affecting communities, especially those of the poor, in several ways: reducing the physical capacities and impairing the emotional development of individuals, increasing the morbidity and mortality risks of children, draining the economic prospects of households, and reducing the development potential of communities.

In recent years rickets has been reported in at least 21 countries. While some of these reports describe sporadic occurrences associated with lifestyle and the lack of solar exposures to support adequate vitamin D biosynthesis, several have described rickets caused by local food systems that fail to provide calcium in adequate amounts and accessible forms. The disease that has been identified in Bangladesh (e.g Cox's Bazaar District) appears to be of the calcium-deficiency type, as has been described in South Africa and Nigeria.[40]

In Bangladesh, children in rickets-endemic areas consume diets deficient not only in calcium but also in iron, zinc, vitamins A and C, riboflavin and protein. Most are stunted; 25-50% show signs of protein-energy and/or riboflavin deficiencies and, in general, they have a significant morbidity burden (malaria, worms, episodic diarrhea). Calcium deficiency is likely to be widespread in Bangladesh, affecting children directly as well as via effects of the health of their care-givers (it is almost certain that most mothers and grandmothers suffer from osteomalacia).[41]

There has been markedly less reported cases in countries located closer to the Equator where children can stay out door naked than other parts of the world, it is also less in nomadic families whose exposure to sunshine is adequate, but that is keeping other factors in balance. A high number of rickets is also observed in the southern Hemisphere's children when they are deprived of fresh air and sunshine.[6]

Children living in lowlands and humid places tend to be victims of rickets, due to the fact that ultraviolet rays may be blocked by the air if it contains dust, soot and various vapours. This explains some facts about the seasonal rickets that is observed in children especially in the pole regions.[6]

Diseases:-

No small part in the emergence of rickets is played by the diseases the child has or has survived. Gastrointestinal disorders lead to disturbance of all forms of metabolism, including that of minerals. In such cases acidosis quickly develops and there is a deficiency not only of vitamin D, but also vitamins A, C and B, which in turn facilitate the development of rickets.[6,24]

Last but not least, most studies have indicated that rickets is a persistent problem in children in the communities and should be suspected in children who present with features of failure to thrive, among other conditions. On the other hand infants and children below five years with rickets have been found to be highly susceptible to various infections and different disease conditions. So in case of improper management of rickets, these may run a very severe course, resulting into their high morbidity and mortality.[6,22]

In summary, poor social economic status of most families, low birth weight and prematurity, unbalanced nutritional diets, short breast-feeding duration, skin colour pigmentation, non exposure to sun light and diseases suffered by both the child and their mothers are among the many major predisposing factors in rickets in children and infants

2.1 Objectives of the study:

General Objective

To identify predisposing factors of rickets in infants and children under five years admitted at Kenyatta National Hospital, Nairobi Kenya.

Specific Objectives:

i) To determine whether parents / guardians of rickets patients have any relevant information regarding the disease and its predisposing factors.

ii) To find out the social demographic characteristics of the guardian / parent(s) of the children with rickets.

iii) To investigate the nutritional history of children with rickets at KNH.

2.2 Materials and methods:

This cross-sectional study was carried out in the paediatrics wards at Kenyatta National Hospital, Nairobi Kenya.

The study population consisted of all the infants and children between 3 months and 5 years of age, who were diagnosed and admitted to the Hospital Paediatric wards with rickets during the study period. There were no exclusion criteria.

The sample size (n) was calculated using the formulae:

$$n = \frac{Z^2 p (1-p)}{d^2} = \frac{1.962 \times 0.6 (1- 0.6)}{0.12} = 92.2$$

d is the degree of precision set at 0.1 (10%)

P is the hypothesized prevalence level (0.6). Risk factors such as prematurity and malnutrition have estimated prevalence of 50-60% in Kenya

Z is the Standard normal deviation set at 1.96 which corresponds to 95%

Ninety four patients were recruited

Authorization from the Kenyatta National Hospital Research and Ethics Committee was granted and the patient's parents / Guardian were asked for consent.

For all the candidates qualifying for entry into the study, files were reviewed every post admission day in the paediatric wards within KNH and then a pre designed questionnaire (appendix iii) was filled with relevant data from the file and the parent or guardian of the patient.

The data collected were transferred from the data collection forms into the SPSS database software for analysis.

CHAPTER 3: RESULTS

3.0 Parents / Guardians knowledge regarding the disease

There were almost equal responses from parents/guardians regarding knowledge of rickets, its causes and its prevention. 48.89% were aware and 51.11% were ignorant. Most of the respondents were females.

3.1 Drugs prescribed to patients

Thirty nine patients out of the ninety four admitted to having had some illnesses before and having taken different medicines but only one was on phenobarbitone, which is implicated in the development of rickets
(Table i).

Table i: Drugs used to treat patients during chronic illnesses

Drug's Name	Frequency	Percentage
Paracetamol	8	13.1
Calcimax (Calcium Supplement)	4	6.6
Septrin (Cotrimoxazole)	4	6.6
Ventolin (Salbutamol)	4	6.6
RHZ (Anti Tb)	3	4.9
X-Pen (Benzyl Penecillin)	3	4.9
Alfa D3 (Cholecalciferol)	3	4.9
Vitamin D2 (Ergocalciferol)	2	3.3
Anti pneumonia drugs	2	3.3
Amoxil (Amoxycilline)	2	3.3
Augmentin	1	1.6
Phenalin (Chlorpheniramine)	1	1.6
Piriton (Chlorpheniramine)	1	1.6
Cough syrup	1	1.6
HZT (Anti Tb)	1	1.6
Calgluconate (Calcium gluconate)	1	1.6
Calmax (Calcium Supplement)	1	1.6
Quinine Syrup	1	1.6
Rocephin (Ceftriaxone)	1	1.6
Phenobarbitone	1	1.6
Amikacin	1	1.6
Bro-Zedex (Mucolytics)	1	1.6
Calpol (Paracetamol)	1	1.6
Alimox (Amoxycilline)	1	1.6
Sulbutamol syrup	1	1.6
Clavulin(Amoxycillin + Clavulonate)	1	1.6
Antimalarial	1	1.6

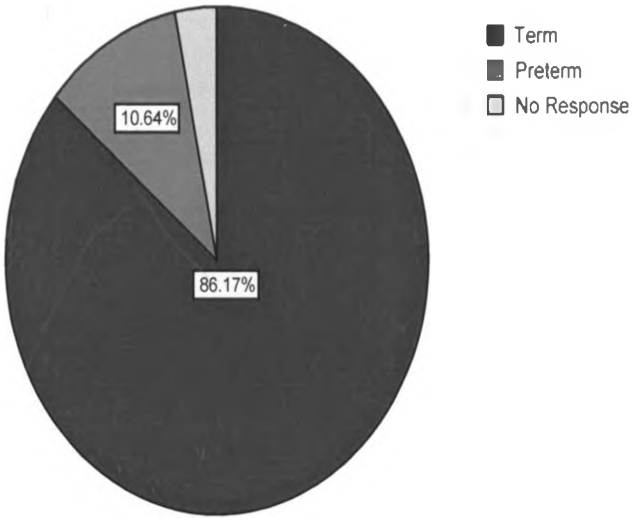
Ceftriaxone	1	1.6
Gentamicin	2	3.3
Zinnat (Cefuroxime)	1	1.6
Rhinathiol (Carbocisteine)	1	1.6
Rifater (Anti TB)	1	1.6
Pyrodoxine (Vit B6)	1	1.6
Ascoril (Expectorant)	1	1.6
Anti Diarrhoea drugs	1	1.6
Total Responses 39	**61**	**100.0**

3.2 Socio-demographic characteristics

3.2.1 Distribution of birth history

In the study, 86.2% were born at term, 10.6% preterm and 3.2% of carers did not know whether the patients were born at term or not (Fig i).

Fig i: The distribution of Birth History



3.2.2 Sex distribution of children

Males accounted for 52.1% while 47.9% were females indicating an almost equal distribution of the disease across the two genders.

3.2.3 Birth weight distribution

This study found that 30.9% of the patients were born below the WHO recommended birth weight of 2500g (Table ii).

Table ii: Birth weight distribution

Interval (g)	Frequency	Percentage
501-1000	3	3.2
1001-1500	3	3.2
1501-2000	8	8.5
2001-2500	15	16.0
2501-3000	28	29.8
3001-3500	29	30.9
3501-4000	5	5.3
4001-4500	2	2.1
4501-5000	1	1.1
Total	**94**	**100.0**

3.2.4 Age distribution

The table shows 85.1% patients were between the age of 3 months and 15 months (Table iii). Only one child was over two years of age (46 months).

Table iii: Age distribution

Class interval (months)	Frequency	Percentage
3.0	4	4.3
3.1-6	24	25.5
6.1-9	27	28.7
9.1-12	17	18.1
12.1-15	8	8.5
15.1-18	6	6.4
18.1-21	4	4.3
21.1-24	3	3.2
45.1-48	1	1.1
Total	**94**	**100.0**

3.2.5 Current body weight distribution

There were two children less than 4 kg and one greater than 10kg. About a half were between 5.5 and 7kg (Table iv).

Table iv: Current body weight distribution

Weight (g)	Frequency	Percentage
3100-3500	1	1.1
3600-4000	1	1.1
4100-4500	4	4.3
4600-5000	6	6.4
5100-5500	10	10.6
5600-6000	12	12.8
6100-6500	16	17.0
6600-7000	12	12.8
7100-7500	10	10.6
7600-8000	10	10.6
8100-8500	5	5.3
8600-9000	3	3.2
9100-9500	1	1.1
9600-10,000	2	2.1
14600-15000	1	1.1
Total	**94**	**100.0**

3.2.6 Child's height distribution

The shortest children were 55-59 cm (6.4%), while the tallest were 80 - 84 cm (5.3%) respectively (Table v). About a half was 60 – 69 cm.

Table v: Child's height distribution

Interval (cm)	Frequency	Percentage
55 - 59	6	6.4
60 - 64	27	28.7
65 - 69	25	26.6
70 - 74	22	23.4
75 - 79	9	9.6
80 - 84	5	5.3
Total	**94**	**100.0**

3.2.7 Distribution of patient's rank in the family

The children were most commonly second born (36.2%), followed by first born (26.6%) and third born (25.5%) (Table vi).

Table vi: Distribution of patient's rank in the family

Rank	Frequency	Percentage
1st Born	25	26.6
2nd Born	34	36.2
3rd Born	24	25.5
4th Born	5	5.3
5th Born	5	5.3
7th Born	1	1.1
Total	**94**	**100.0**

3.2.8 Child's age at first dentition

In this study, 46.8% of the children below 10 months had no teeth, while 34% had (Table vii). Only 6.4% did not have their first dentition after 10 months.

Table vii: The child's age at first dentition

Age	With teeth		With no teeth		Total
	Frequency	Percentage	Frequency	Percentage	
10 Months and below	32	34.0	44	46.8	76
Above 10 Months	8	8.5	6	6.4	14
Don't remember	4	4.3			4
Total	**44**	**46.8**	**50**	**53.2**	**94**

The six who failed to have dentition by the end of the expected had the following characteristics:

The oldest patient among those who failed to develop teeth by ten months was 20 months and the youngest were 12 months. Majority had their body weight below 60% of the expected body weight (Table viii).

Table viii: Patients who failed to develop teeth at ten months

sex	Age(mo)	Birth term	Rank in the family	Expected body weight (%)
M	20	preterm	2nd born	60 - 80
F	19	preterm	3rd born	< 60
M	17	term	1st born	< 60
F	13	term	1st born	< 60
F	12	term	1st born	< 60
M	12	term	3rd born	< 60

3.2.9 Prevalence of twin patients

In the study, seven pairs of twins were among the patients and also included three members of one family aged 4years and was considered, the other siblings were 8 and 12 years respectively.

3.2.10 Expected normal body weight distribution

Many patients had their body weight within the range of 60 – 80% of age and height related average weight (Table ix). The 18.1% had their body weights below 60% an indication of very poor health status.

Table ix: Child's expected normal body weight distribution

Body weight	Frequency	Percentage
Below 60%	17	18.1
60-80%	40	42.6
80 % & Above	36	38.3
Total	**93**	**99**

3.2.11 Birth spacing distribution

Twenty six percent of the patients were first born and were presumed to be last born too (Table x).

Table x: Child's birth spacing distribution

Status of the child	Frequency	Percentage
1st born with no siblings	25	26.6
1st born with siblings	0	0
Last born with previous > 2years	51	54.3
Last born with previous sibling < 2years	11	11.7
Middle born previous & next >2 years apart	6	6.4
Middle born with previous < 2years apart	1	1.1
Total response 94	**94**	**100.1**

3.2.12 Distribution of previous illnesses

It was observed that 41.5% of the patients had illnesses previously compared to 58.5% who did not (Table xi).

Table xi: Distribution of any previous illness to the child

Previous illness	Frequency	Percentage
Yes	39	41.5
No	55	58.5
Total	**94**	**100.0**

3.2.13 Illnesses at admission

All the patients with rickets had had other conditions like pneumonia (60.6%), PEM (16.0%), diarrhoea (8.5%), among others (Table xii). Most patients had more than one illness.

Table xii: Illnesses diagnosed at admission

Disease/Symptom	Frequency	Percentage of cases
Pneumonia	57	60.6
Protein Energy Malnutrition (PEM)	15	16.0
Meningitis	11	11.7
Gastroenterititis	8	8.5
Diarrhoea	8	8.5
Severe dehydration	7	7.4
Vomiting	7	7.4
Convulsions	6	6.4
Jaundice	4	4.3
Failure to Thrive (F.T.T)	1	1.1
Injury Trauma	1	1.1
Rachitic chest problem	2	2.1
Cerebral palsy	1	1.1
Cardiac problem	1	1.1
HIV Exposed	2	1.2
Urinary Tract Disease (URT)	2	1.2
Perinatal abscess	1	1.1
Tuberculosis	3	3.2
Fever	3	3.2
Malaria	3	3.2
Common Cold	3	3.2
Bronchitis	4	4.3
Anaemia	1	1.1
Dermatitis	1	1.1
Arthritis	1	1.1
Neonatal sepsis	1	1.1
Genetic disorder	1	1.1
Total responses in 94	**160**	

3.2.14 Previous illnesses

Among the 94 patients, thirty six had had previous illnesses or signs and symptoms that included 44.7% of Pneumonia, 23.7% rickets, 21.1% Fevers, 18.4% Tuberculosis and coughing respectively (Table xiii). The rest are shown below.

Table xiii: Previous illnesses / conditions suffered by the patient

Previous condition / illness	Frequency	Percentage of reported illness
Pneumonia	17	44.7
Rickets	9	23.7
Fever	8	21.1
Coughing	7	18.4
Tuberculosis	7	18.4
Malaria	3	7.9
Meningitis	3	7.9
Sweating	1	2.6
Continuous Cold	2	5.3
Asthma	1	2.6
Jaundice	2	5.3
Diarrhoea	2	5.3
Vomiting	2	5.3
Gastroenteritis	2	5.3
Dermatitis	1	2.6
Arthritis	1	2.6
Genetic Disorder	1	2.6
Total responses in 36 patients	**69**	

3.2.15 Mother's illnesses during pregnancy

Only 21.28% of mothers reported at least one illness during gestation.

3.2.16 Mother's post partum illnesses

8.5% of the mothers admitted to have been ill during breast feeding period (Table xiv).

Table xiv: Distribution of illnesses of the mother during breast feeding

Illness after child birth	Frequency	Percentage of mothers
Yes	8	8.5
No	85	90.4
No response	1	1.1
Total responses	**94**	**100.0**

3.2.17 Status of the parents and household head

Most mothers (98.9%) of the children and infants in this study were alive, only one had died, and most fathers were still alive (97.8%), only two had died.

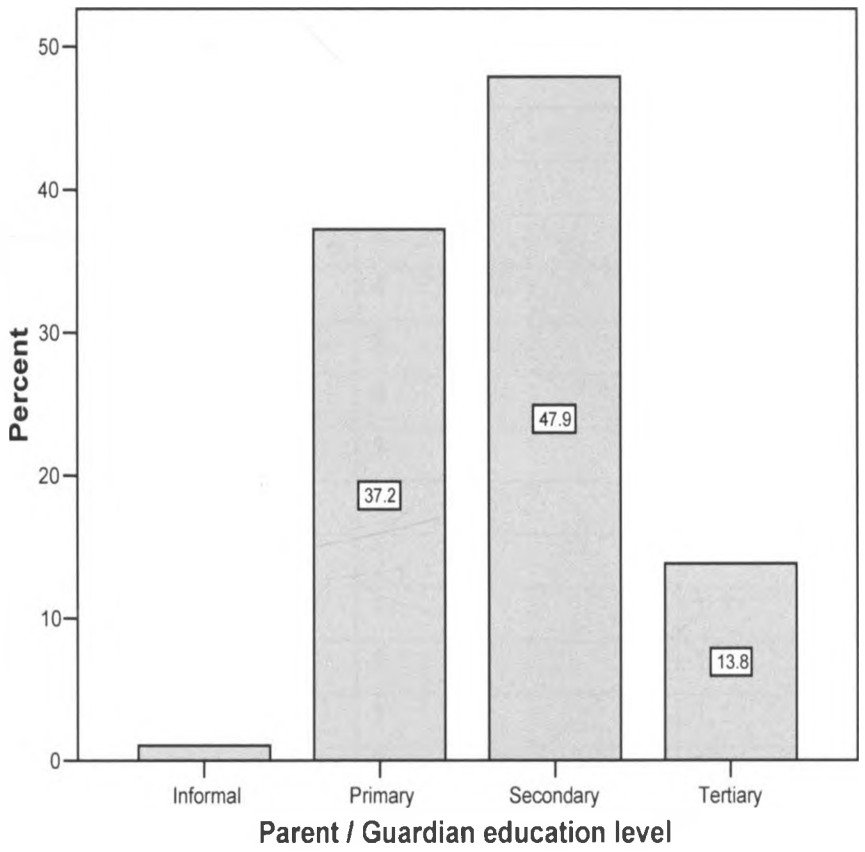
All children had at least one parent still alive.

Most households (73.4%) were headed by males and 25.5% by females. Less than 2% had single parents.

3.2.18 Education and occupation of the parents / guardian

61.7% of the children had a carer who had at least completed secondary school level of education and tertiary institutions, with less than 2% of the carers having only informal education (Fig ii). This question related only to carers who accompanied the patients in the hospital.

Fig ii: Parent / Guardian education level



Among the respondents 25.5% were house wives with no regular income at all, 27.7% were running small businesses in their localities, 9.6% were casual labours, 6.4% were small farmers and the rest were doing very low income jobs (Table xv). This was according the respondents or carers.

Table xv: Parents / Guardians occupations

Occupation	Frequency	Percentage
House wife/No Job	24	25.5
Small Business	26	27.7
Casual	9	9.6
Farmer	6	6.4
Clerk	2	2.1
Mechanic	1	1.1
College Student	2	2.1
Diploma in Dental medicine	1	1.1
Technologist	1	1.1
Work on Contracts	2	2.1
Care Taker	4	4.3
Tailor	1	1.1
Journalist	3	3.2
Security Man	1	1.1
Electrician	1	1.1
Conductor	1	1.1
Teacher	1	1.1
Carpenter	1	1.1
Driver	1	1.1
Receptionist	2	2.1
Pastor	1	1.1
Mason	1	1.1
No response	3	3.2
Total	**94**	**100.0**

3.2.19 Relationship of care givers to the child

A big number of patients (75.5%) were taken care of by their mothers, 13.8% by house girls and mothers partly and a handful of them by their fathers, aunt's and baby care centres as indicated below and about 22 were taken care by more than one (Table xvi).

Table xvi: Distribution of the relationship of the children caregiver

Relationship	Frequency	Percentage
Mother	71	75.5
Father +Aunt	3	3.2
House girl + Mother	13	13.8
Aunt	4	4.3
Baby Care Centre + Father + Aunt	3	3.2
Total responses	**94**	**100**

3.2.20 Parents / guardians religion

The distribution of the parent's or guardian's religion indicated that over 97% were Christians and only two were Muslims.

3.2.21 Status and periods of sun bathing

Patients who sun bathed were about 34% while about 62.8% did not for one reason or another (Table xvii).Here sunbathing was defined as taking children and infants purposely with a reason to obtain vitamin D.

Table xvii: Status of sun bathing with minimum clothing

Status of sun bathing	Frequency	Percentage
Yes	32	34.0
No	59	62.8
No response	3	3.2
Total responses	**94**	**100.0**

Sixty two percent of the patients were not sun bathed at all, and about 21.3% of the children were sun bathed during the morning (Table xviii).

Table xviii: Periods of the day that children were sun bathing

Period	Frequency	Percentage
8:00 – 12:00 Morning	20	21.3
12:01 – 2:00 Mid day	1	1.0
2:01 – 4:00 afternoon	6	6.4
4:01 – 6:00 evening	1	1.0
morning and afternoon	4	4.3
No response	3	3.2
Did not sun bath	59	62.8
Total responses	**94**	**100.0**

3.2.22 Districts of origin

It is evident that most of these patients had their ancestral origins from the central province and Nairobi, led by Muranga North (14.9%), Kiambu (10.6%), Nyeri (9.6%) , Nairobi (9.6%) and the rest were from different parts of Kenya (Table xix).

Table xix: Patient's ancestral Home Districts

Ancestral Home District	Frequency	Percentage
Nairobi	10	10.6
Thika	3	3.2
Nakuru	5	5.3
Moyale	1	1.1
Kiambu	10	10.6
Kajiando	2	2.1
Mumias	1	1.1
Kitui	4	4.3
Kirinyaga	2	2.1
Murang'a North	14	14.9
Machakos	5	5.3
Nyandarua	5	5.3
Murang'a South	1	1.1
Matuu	1	1.1
Embu	4	4.3
Vihiga	1	1.1
Kangundo	1	1.1
Nyeri	9	9.6
Makueni	3	3.2
Kitale	1	1.1
Samburu	1	1.1
Busia	1	1.1
Laikipia	1	1.1
Bondo	1	1.1
Bungoma	2	2.1
Siaya	1	1.1
Meru	1	1.1
Nyambene	1	1.1
Migori	**1**	**1.1**
Not known	**1**	**1.1**
Total responses	**94**	**100.0**

3.2.23 Status of covering the head among mothers

During the study, 24.5% of the mothers were found with head scarf (Table xx).

Table xx: Mothers style of wearing clothes especially head covering

Covers Head	Frequency	Percentage
Yes	23	24.5
No	68	72.3
No response	3	3.2
Total responses	**94**	**100.0**

3.2.24 Family history of rickets

16% of the parents / guardians admitted to have had rickets in their families (Table xxi). This was defined by the patient's carer by citing that one or more close relative(s) in their families have had rickets.

Table xxi: Family history of rickets

Any History	Frequency	Percentage
Yes	15	16.0
No	78	83.0
Don't know	**1**	**1.1**
Total responses	**94**	**100.0**

3.2.25 Financial ability of the family

About 58.6% of the parents /guardians said that it would be difficult or even not possible to clear the hospital bills of their patients (Table xxii).

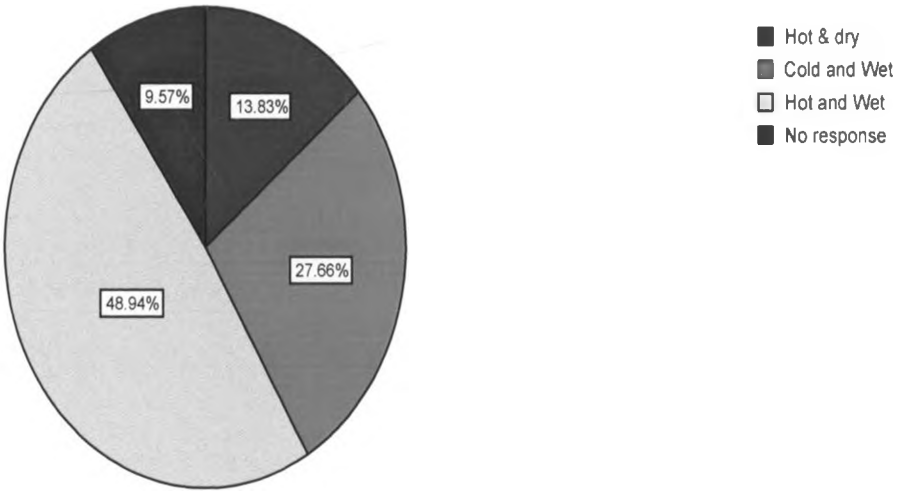
Table xxii: Ability of the patient's family to clear the medical bills

Ability to clear hospital bill	Frequency	Percentage
Easy	35	37.2
Difficult	40	42.6
Not able	15	16.0
No response	4	4.3
Total responses	**94**	**100.0**

3.2.26 Climatic conditions of area of residence

Generally patients came from climatic conditions that were hot and wet most of the year (54.1%) and about 30.6% from cold and wet regions respectively (Fig iii). The areas climatic conditions were defined by patient's carers.

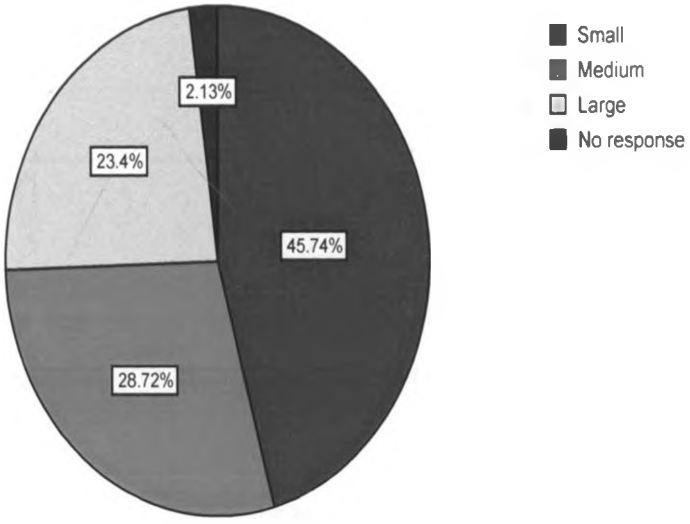
Fig iii: Climatic conditions of area of residence



3.2.27 Family compound size

From the study, about 46.7% of the families had very limited space in their compounds and 29.3% with medium size compounds respectively (Fig iv). The compound size was defined by whether it had enough space where child would be sunbathed, play and sit. This was defined by the carers assisted by the investigator.

Fig iv: Family compound size



3.3 Family Nutritional background

3.3.1 Duration of Exclusive Breastfeeding

A large number of patients (77.7%) were exclusively breast fed for less than six months (Table xxiii).

Table xxiii: The child's Duration of EBM distribution

Age in Months	Frequency	Percentage of children
Below 6 months	73	77.7
6 months & Above	21	22.3
Total responses	**94**	**100.0**

3.3.2 Weaning age

The data below shows that about 60% of the patients were introduced to solid food stuffs before the age of six months and about 2% below one month (Table xxiv).

Table xxiv: Weaning age

Age in Months	Frequency	Percentage of children
Below 1 Month	2	2.4
1.00	4	4.3
1.50	1	1.1
2.00	8	8.5
2.50	1	1.1
3.00	9	9.6
4.00	19	20.2
4.50	2	2.1
5.00	11	11.7
6.00	19	20.2
7.00	4	4.3
8.00	2	2.1
9.00	1	1.1
Don't remember the age at which solid food introduction	11	11.7
Total response	**94**	**100.0**

3.3.3 Frequency of feeding

About 23.4% of the patients had feeding frequencies of 2 or less per day while 72.3% fed three or more times in a day (Table xxv).

Table xxv: Feeding habits

No of meals per day	Frequency	Percentage of all children
=/< 2 times a day	22	23.4
=/> 3 times a day	68	72.3
No Response	4	4.3
Total response	**94**	**100.0**

3.3.4 Food stuffs given during weaning and after stoppage of breast feeding

Types of food given to the patients were 20.2% Porridge, 16.3% milk, 8.7% Pumpkins, 8.3% fruits, 7.9% Bananas and after stoppage of breast feeding, the main meals were Bananas (15.1%), Pumpkins (15.1%), Porridge (13.3%), Potatoes (10.8%), Spinach (7.9%) and Milk (7.9%) , the rest as shown below (Table xxvi).

Table xxvi: Food given during weaning and after stoppage of breast feeding

Name of food item	Frequency at weaning	Percentage at weaning	Frequency at stoppage	Percentage at stoppage
Porridge	51	20.2	37	13.3
Milk	41	16.3	22	7.9
Pumpkins	22	8.7	42	15.1
Fruits	21	8.3	11	3.9
Bananas	20	7.9	42	15.1
Potatoes	16	6.3	30	10.8
Pawpaw	16	6.3	8	.4
Spinach	14	5.6	22	7.9
Water	11	4.4	--	--
Soup	2	.8	4	1.4
Sweet potatoes	1	.4	1	.4
Water melons	1	.4	2	.7
Ripe Bananas	3	1.2	--	--
Passion juice	3	3.7	2	.7
Oranges	1	.4	3	1.1
Pineapples	1	.4	--	--
Avocados	1	.4	1	.4
Nan pellargon	4	1.6	--	--
Weetabix	3	1.2	3	1.1
Celerac	3	1.2	--	--
Ugali	5	2.0	13	4.7
Sukuma	2	.8	--	--
Tomatoes	2	.8	1	.4
Carrots	3	1.2	5	1.5
Beans	3	1.2	9	3.2
Green grams	2	.8	5	1.8
Milk formula	--	--	2	.7
Rice	--	--	6	2.2
Meat	--	--	1	.4
Soup	--	--	4	1.4
Amaranthus	--	--	3	1.1
Total responses	**252**	**100.0**	**279**	**100.0**

3.3.5 Food taken during gestation period and after delivery

During pregnancy mothers largely fed on cereal based food stuffs like Ugali (71.7%), Rice (56.5%), Githeri (40.2%) and very few food stuffs were from animal sources and the main food(s) used by mothers after delivery were Ugali (66.7%), Rice (51.9%), Githeri (33.3%) and Porridge respectively (Table xxix). There were a few animal source foods like meat at (24.7%), Fish (11.1%) and milk (14.8%), (Table xxvii).

Table xxvii: Main Meals taken by patient's mothers during Pregnancy

Food item Name	Frequency	Case Percentage	Frequency	Case Percentage
Ugali	66	71.7	54	66.7
Rice	52	56.5	42	51.9
Githeri (Maize+Beans)	37	40.2	27	33.3
Sukuma wiki	28	30.4	22	27.2
Beans	26	28.3	18	22.2
Fruits	24	26.1	12	14.8
Meat	20	21.7	20	24.7
Milk	15	16.3	12	14.8
Porridge	15	16.3	27	33.3
Cabbage	15	16.3	18	22.2
Green bananas	13	14.1	11	13.6
Green grams	13	14.1	9	11.1
Spinach	12	13.0	8	9.9
Potatoes	10	10.9	3	3.7
Fish	10	10.9	9	11.1
Mukimo	2	2.2	3	3.7
Tea	7	7.6	3	3.7
Soda	3	3.3	--	--
Soup	3	3.3	14	17.3
Liver	4	4.3	1	1.2
Chicken	1	1.1	--	--
Mutura	1	1.1	--	--
Eggs	4	4.3	2	2.5
Oranges	3	3.3	--	--
Pawpaw's	2	2.2	--	--
Avocado	1	1.1	--	--
Mangoes	4	4.3	--	--
Passion fruits	4	4.3	--	--
Banana fruits	2	2.2	--	--
Water melon fruits	1	1.1	--	--
Chapattis	6	6.5	10	12.3
Chips	4	4.3	2	2.5
Sweet potatoes	4	4.3	3	3.7
Arrow roots	1	1.1	1	1.2
Njahi	5	5.4	16	19.8
Pigeon peas	1	1.1	9	11.1
Omena	2	2.2	3	3.7

Pumpkins	1	1.1	--	--
Bread	3	3.3	--	--
Spaghetti	--	--	1	1.2
Amarathus	--	--	1	1.2
Mirenda	--	--	2	2.5
Sagaa	--	--	2	2.5
Juice	--	--	1	1.2
Yams	--	--	1	1.2
Total responses in from 92 & 81 respectively	**425.0**		**367**	

3.3.6 Craving for food

A big percentage of mothers (87.2%) craved for particular types of food items and soil (Table xxviii). This is an indication of poor nutrition among mothers.

Table xxviii: Distribution of mothers who craved for particular food stuffs

S.No	Craved for food	Frequency	Percentage
1	**Yes**	82	87.2
2	No	11	11.7
3	No response	1	1.1
Total	**responses**	**94**	**100.0**

3.3.7 Foods craved for during gestation

Of the eighty four respondents, the main food stuffs craved for by mothers during pregnancy were: Githeri (20.2%), Rice (19.0%), Ugali (16.7%), Milk (15.5%), Soil (14.3%) and fruits respectively, also some animal source foods like meat (9.5%), eggs (14.3%), matumbo (6.0%) (Table xxix).

Table xxix: Foods Craved for by mothers during pregnancy

Food Craved for	Frequency	Case Percentage
Githeri	17	20.2
Rice	16	19.0
Ugali	14	16.7
Milk	13	15.5
Fruits	12	14.3
Stones (Soil)	12	14.3
Soda	9	10.7
Meat	8	9.5
Sukuma	6	7.1
Bananas	5	6.0
Chips	5	6.0
Fish	5	6.0
Beans	5	6.0
Kienyenji	4	4.8
Eggs	4	4.8
Mangoes	4	4.8
Liver	4	4.8
Leafy green vegetables	4	4.8
Sweet potatoes	4	4.8
Sugar	3	3.6
Muthokoi	1	1.2
Groundnuts	1	1.2
Soybeans	1	1.2
Njahi	1	1.2
Green	2	2.4
Pigeon peas	2	2.4
Pumpkins	1	1.2
Passion fruits	2	2.4
Avocados	1	1.2
Pineapples	1	1.2
Cabbages	3	3.6
Chicken	1	1.2
Roasted meat	2	2.4
Matumbo	2	2.4
Mutura	2	2.4
Carrots	1	1.2
Potatoes	3	3.6
Arrow roots	2	2.4
Chapattis	1	1.2

Tea	2	2.4
Cultured milk	2	2.4
Soup	3	3.6
Oranges	1	1.2
Total responses = 84	**200**	

3.3.8 Food items avoided during gestation

Among the eighty respondents, Ugali was avoided most (26.3%), followed by sukuma (21.3%), meat and rice each (17.5%), cabbages (12.5%) (Table xxx). Fruits were the least avoided.

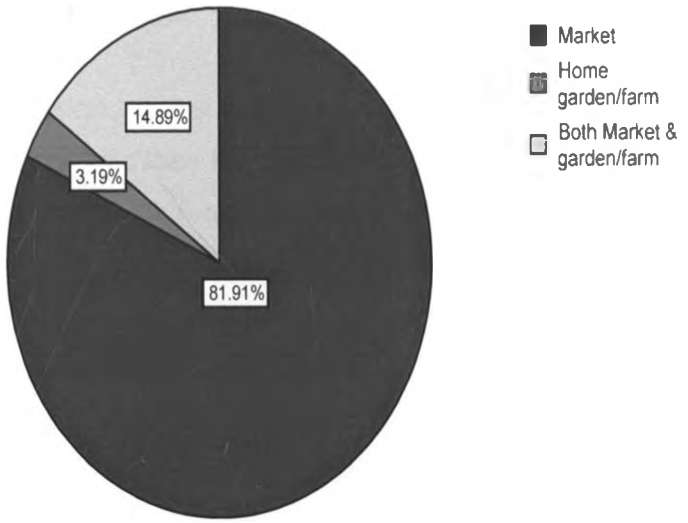
Table xxx: Food items avoided by Mothers during pregnancy

Food item Name	Frequency	Case Percentage
Ugali	21	26.3
Sukuma	17	21.3
Rice	14	17.5
Meat	14	17.5
Cabbages	10	12.5
Beans	8	10.0
Eggs	7	8.8
Tea	7	8.8
Chapattis (Cooked with yellow fat)	7	8.8
Porridge	7	8.8
Githeri	6	7.5
Green grams	5	6.3
Bananas	4	5.0
Milk	3	3.8
Fish	3	3.8
Omena	1	1.3
Chips	2	2.5
Traditional vegetables (Amaranth)	1	1.3
Soup	2	2.5
Sweet potatoes	2	2.5
Irish potatoes	2	2.5
Cassava	2	2.5
Spinach	2	2.5
Bread	2	2.5
Mukimo	1	1.3
Matumbo	1	1.3
Peas	1	1.3
Spaghettis	1	1.3
Avocados	1	1.3
Oranges	1	1.3
Pawpaws	1	1.3
Total responses = 80	**157**	

3.3.9 Families source of food

Around 82% of the patient's families used market as their sole source of food items and 14.89% got their food items from both market and gardens (Fig vi). A negligible 3.19% were able to get food solely from home gardens.

Fig v: Source of food (s)



CHAPTER 4: DISCUSSION, CONCLUSION AND RECOMMENDATIONS

4.1 Discussion

The predisposing factors of rickets in the 94 infants and children in this study were found to be interrelated and comparable to many other studies done in Nyeri, Embu, KNH,[5] and Machakos in Kenya,[32] Bangladesh,[3] China,[1] England, [14] Egypt, Ethiopia,[46] Iran,[28] Jordan, [31] Melbourne Australia, [30] Nigeria,[40] Russia. [6].

4.1.1 Knowledge about the disease

Among the many predisposing factors, poor understanding of the disease amongst the parents / guardians and communities had a crucial role in the prevalence of rickets. The study comprised of more than 98% females whose difference in responses as to whether they had knowledge or not regarding rickets, was minimal. They knew the name "Rickets" but not its causes / risk factors or how it can be prevented and managed.

All these parents / guardians brought the patients to the hospital (KNH) with complaints of some clinical symptoms like: poor feeding, diarrhoea, vomiting, and fever, hotness of the body, coughing, poor weight gain and other childhood manifestations of being unwell.

Only about 16% were readmissions with rickets and even these presented with another complaints. We can presume the readmission parents had a prior idea of what rickets was but the rest were informed after diagnosis on their admission. Most of them had no knowledge about the disease before. Poor knowledge about the disease is rated among the highest predisposing factors to rickets as seen even in other studies done in Ethiopia.[42,43] Religion, occupation, educational status and residence (village) of the mothers / care givers were among the cited predisposing factors. Other major causes of nutritional rickets in Ethiopian children were found to be lack of exposure to sunshine and / or inadequate intake of vitamin D. Lack of awareness and traditional beliefs were major causes for not exposing infants to sunshine.

4.1.2 Drug involvement

Drugs are among predisposing factors of rickets especially anticonvulsant drugs such as phenobarbitone which causes defective metabolism of the parent vitamin D to active metabolites.[22,24] In one study carried out in the Paediatric Department of Athens University, Athens, Greece, twenty-five patients (49%) on anticonvulsants, acquired hypovitaminosis D during the study period.[26] This study did not support the claim as only one patient was on

anticonvulsants; phenobarbitone for that case. Some studies have indicated hypovitaminosis D in patients on prolonged phenobarbital.[44]

4.1.3 Sociodemographic Characteristics

In this study besides so many predisposing factors that were involved in the aetiopathogenesis of this disease, poor social economic status of the families of the patients had a major role to play. In other studies carried out in Bangladesh, children in rickets-endemic areas consumed diets deficient not only in calcium but also in iron, zinc, vitamins A and C, riboflavin and protein. Most were stunted; 25-50% showed signs of protein-energy and/or riboflavin deficiencies and, in general, they had a significant morbidity burden (malaria, worms, episodic diarrhoea).[37,41] Research carried out by the Institute for Social and Economic Research (ISER) showed a relationship between poverty in childhood and well-being of adults, demonstrating that child poverty can leave a damaging long-term legacy, rickets included.[21,37] The diets of people on low incomes in Bangladesh were often nutritionally poor, relying on 'cheap calories' from processed low-cost food. NCH's study found that mothers sometimes went without food to meet the needs of their children. A Barnardo's study found that this was especially true in the school holidays when families were managing without free school meals.[37,39]

The poverty levels in the families of the patients in this study were reflected by the occupation and activities that most families were engaged in, the kind of food stuffs the families fed on and on the their responses regarding the ability of these families clearing hospital bills for their patients where about 65% of them said they were not able or sure how to pay the bills. Most families were still young which could also imply inability to have accumulated enough wealth to smoothly run their daily expenses. Also they may not have inherited anything tangible from their families which in most cases were facing the same financial constraints as those who were still alive.

Little or non exposure of infants and children in the sun for ultraviolet light has an important role in the development of rickets in the study group, since non exposure leads to limited or non biosynthesis of adequate vitamin D through endogenous production.[33,42,45] Although vitamin D deficiency is accepted as the basic problem underlying the disease, others postulate that a deficiency of dietary calcium, rather than vitamin D, is often responsible for the nutritional rickets in sunny countries.[43] The fact that there was no difference in the dietary calcium intake between cases and controls and the

significant difference observed between case and controls in terms of frequency of exposure to sunlight per week reflected that vitamin D deficiency emanating from poor exposure to radiant energy was the main cause of rickets in Ethiopian children.[42,46]

Lack of vitamin D is caused by problems with our diet and lifestyle. Most of our vitamin D is made by the body itself when sun shines directly onto the skin. People can lack vitamin D if they spend too much time indoors, or cover themselves up when outside. The problem is worse in cold climates and for people with dark skin where the effect of the sun on the skin is less.[43,46]

Basking in the sun for 15 minutes three times weekly, with hands, arms and face uncovered, is said to be enough for fair-skinned people. Darker-skinned people will need more sunshine and longer periods of exposure.

Due to the social demographic settings of most of their families and to some extent cultural beliefs, some of the infants and children were never taken out in the open: Therefore the abundance of sunlight in the country was not optimally utilized. By and large these children were always kept indoors and a few that went out once in a while would be heavily dressed and their heads covered for the fear of getting attacked by winds and strong sun according to the parents/ carers. This particular situation left the children with no option for any other source of the highly needed vitamin D, calcium and phosphorus, apart from the poor non nutritious food stuffs available to them

From the responses 62.2% said they were sun bathing their children, but among these only 21.3% seemed to have sun bathed within the recommended range of time 8am – 12 noon, and still would be heavily clothed during that time. 34% of the respondents never took their children outside for the purpose of sun bathing. These irregularities and inconsistencies in sun bathing contributed to lack of vitamin D leading to development of rickets in the patients.

Most of the patient's families came from Central Kenya and Nairobi; these two distinctive areas are assumed to have the highest levels of income, health facilities and other infrastructures in the country. The role of socio-cultural taboos and beliefs in the whole issue of this disease was not investigated in this study, but was found to be important in neighbouring Ethiopia.[42]

Admissions were commonly from: Muranga North with 14.9%, Kiambu 10.6%, Nyeri 9.6% and Nairobi 9.6%. This may be due to the fact that the mentioned areas have high populations in relation to the rest of the country, but also may be a reflection of some socio

cultural practices. Proximity and easy accessibility of the regions to the Kenyatta National Hospital might too have had a role in the high prevalence of rickets among patients from the two regions at this facility. Due to poor socio economic status of the families, most patients were first treated on herbs from their local herbalists who offer cheap service in relation to western medicine and it is only after a long wait and detritions of patient's health that their parents sought help from modern medical services. Some of the patients were locals from Nairobi area but most were from outside the city, due to the high quality services offered and relatively low costs at the facility in its category, according to the carers. In a similar study carried out in rickets-endemic areas of Bangladesh (e.g. Chakaria) it was found that the victims were not necessarily from among the poorest areas of Bangladesh and the at-risk children were not among the most severely malnourished children in the country. This raised several questions that demanded an answer,[37,41] (see last paragraph of chapter 2).There is not just one factor involved in the pathogegenesis of the disease but multiple factors are at play. Those who are poor or poorly-educated may not make best use of health resources, despite having greater need.

Of the total respondents,16% admitted to have had rickets in their families in the past, suggesting a possibility of some kind of familial rickets in Kenya. Whether the 16% was familial rickets or not was not confirmed, but involvement of other predisposing factors may not be ignored. In other studies done in UK rickets has been sporadic.[14] Surveys of vitamin D sufficiency in the United Kingdom showed that 25% of children of South Asian origin start winter with low vitamin D stores as assessed by measuring 25-hydroxy vitamin D in serum.[33] In another survey, 480 households were investigated and the ratio of non-rickets to rickets households was 1:4 in parts of Bangladesh.[3]

In Kenya there are no inhabited areas with climatic conditions that would favour development of rickets. Although there some cold spells around the central and Nairobi areas around mid year, they are not extreme. There is sufficient sun, with no extreme humidity, so other predisposing factors were to blame.

It came out clearly that most families did not have enough space at home, as most lived in slum areas with congested structures, and a few in high rise buildings. Those who had enough space especially in the rural areas did not exposure their children because of fear of domestic animals like dogs and some mistrust among neighbours along with other cultural taboos. Compound size was another subject discussed as far as children's movement and restriction was concerned, including the spaces where mothers / guardians and care takers could have enough space to take children to play or even sun

bathe. 45.74% had a very small compound, 28.72% were medium size and 23.4% large. Carers assisted by the investigator, defined the size of the compound in relation to having space where children can go for sunbathing, playing and sitting with convenience.

Among the other issues studied was, whether the study subjects were born at term (at least 37 weeks of gestation) or preterm (before 37 week). A previous study at KNH found that premature babies had a prevalence of rickets of 58%[5] but this study found that only 10.6% of rickets children were born prematurely which indicates that prematurity alone cannot be blamed for this disease without the involvement of other risk factors.

Gender of the patients was cited by some studies which showed that males were more likely to have rickets than females by a ratio of 1.6:1 to 1.2:1 male: female and even more.[5,23,43] This study showed no significant difference between the two genders, since the ratio was 1.14: 1 male to female.

Low birth weight is a predisposing factor to rickets. According to previous research, children and infants having birth weight of 2000g and below have high chances of developing rickets.[5] Surprisingly in this study only 13% of the rickets victims were born with weight of 2000g and below, and about 69% were 2500g and above which is the WHO recommended weight. Therefore, this finding suggests that birth weight alone can not prevent rickets in the presence of other risk factors.

In this study we observed that about 18% of the patients had body weights associated with poor survival with the inference that their future was hanging in a balance of between life and death. This group had their body weight below 60% of the expected weight and this revolved around mostly nutritional issues, past and present illnesses as well as severe dehydration caused by diarrhoea, vomiting and related factors comparable to other studies.[43,48]

The children in this study generally had a short stature and stunted growth in regard to what was expected in individuals with normal body growth and development. This feature leaves the researcher with no doubt on the disastrous effect of rickets on the growing bones and the skeletal systems of these young growing infants and children. Previous studies showed that even their mental capacities including school performance and abilities to learn were impaired.[27,39]

About 87.2% of the patients in this study were 1st, 2nd and 3rd born. The high levels of unemployment and poverty in many young families in the country, especially in urban suburbs and slums leaves the children from these struggling families with no proper parental care as both parents tend to run around looking for what would make ends meet. This exposes them to various harsh and unbearable conditions resulting in rickets and much other disease. It was found predominant (87.2%) in second born and above in a study done in Amman - Jordan[31] while it was 73.4% in second born and above in this study.

Patient's age played a major role. 85% were aged 3 months to 15 months and the prevalence was highest at 6 months, at the time when they started sitting without support and when most were weaned to solid foods. This is when children were increasingly active. Also at 9 months, they started running around and standing, putting pressure on the body that might not have been well nourished due to lack of adequate nutritional requirements at the time when they were needed most.

Among all the parents, 61.7 % had at least completed secondary school education, with less than 2% with informal education. Basically parents or guardians might have lacked proper knowledge on the disease, and other contributing factors might have played a significant role in the development of rickets. With this finding we can confidently rule out the level of education as the predisposing factor but the knowledge on the disease was likely to blame. A study in Nigeria, described lack of western education as one of the predisposing factors.[49]

In previous studies late dentition (first teeth erupting after 10 months of age) was taken as one of the signs of rickets.[1] However in this study only 6.4% did not have teeth in the expected time, the oldest was 20 months while the youngest were 12 months. Suggesting this is not among the cardinal criteria for diagnosing rickets.

Poor birth control and child spacing have been found to be important predisposing factors to rickets in previous studies.[39] In this study, spacing of 2 years or more between the patient and the nearest sibling carried a risk of about 54.3% and 11.7% for prior and subsequent siblings respectively. This finding was important because in these poor social economic conditions poor families would tend to distribute the little resources available amongst the new born and other members in the family. They also tend to give more care to the new born assuming the older child is not in need of more attention and care.

When it comes to the role played by previous or current diseases that the patients have had, rickets like any other childhood disease seem to compromise the body's immune system such that many infectious diseases can easily attack the victim. Pneumonia was common, as in other previous studies.[31,39] Efforts to prevent vitamin D or calcium deficiency may result in significant reductions in morbidity and mortality.

Common concurrent diseases included pneumonia (60.6%), protein energy malnutrition (16%), meningitis (11.7%), diarrhoea (8.5%), vomiting (7.4%) and severe dehydration (7.4%). These conditions and many others impaired the patient's ability to feed well, making any movement to the outside for sun bathing in search of ultraviolet light difficult and hence worsening the whole situation to the extent that even their parents who had been providing for them would keep at home or in the hospital nursing them.

The situation above was almost similar to when their mothers have had illnesses during pregnancy and after giving birth. In this study 21% and 8.5% of mothers admitted to have had at least one illness during and after pregnancy respectively. Therefore some of the children and infants had their mother's ill health condition together with other predisposing factors play a role in the aetiopathogenesis of rickets as seen in previous studies.[6,17]

The gender of the household head and life status were not significant contributors. This is because above 95% of the children and infants had both parents living and staying together, while over 73% of the households were headed by males, playing the role of bread winners. So this is where the question of severe poverty amongst families remains the ultimate player and major predisposing factor in these children and infants with rickets as seen in other studies.[41,46]

Mothers being the best custodian of their children, this indicates that rickets in these children was either due to other factors that parents or guardians especially mothers could not contain.

About 75% of the patients were taken care of by their mothers, 13.8% by house girls and mothers together and only a handful of them by their fathers, aunt's and baby care centres. So it is evident that despite mothers being keen to take care of their young ones, it did not stop these children and infants from getting rickets when the predisposing factors implicated were at play.

Muslim Women's culture of covering their heads can lead to limited access and exposure of their heads to ultraviolet light hence inhibition of vitamin D synthesis. About 24% who were

not Muslims admitted to be fond of covering their heads. This may be associated with covering their children too, especially girls.

Nutritional aspects

Children with only a few days to months of exclusive breastfeeding and early interruption of breastfeeding with almost zero additional essential minerals and vitamins might have greater risks of the disease.[20,46] About 77.7% of the patients in this study were exclusively breast fed for less than the 6 months recommended by the World Health Organization. On the other hand breast feeding alone, especially in black mothers would not be enough to supply the required nutrients and mineral quantities without external supplementation of some minerals like phosphorus, calcium and vitamin D to prevent the occurrence of rickets in those at high risk.[50]

On top of all the nutritional shortfalls, these patients and their families lack the food stuffs used as supplements during weaning periods and more than 90% were on cereal based meals. There were little vegetables and fruits, with almost no animal source food stuffs, like fish, meat, eggs, milk and others of importance to make a balanced diet. This translated into serious shortage of body building nutrients (proteins) especially of animal source, minerals and vitamins which are responsible for proper body growth and nourishment.[40,46]

Surprisingly the food stuffs which the patients were feeding on were of the same poor quality and non-nutritious grades as their mothers. This is an indication of underlying real danger posed by the status of poor nutrition in families and communities with very low social economic levels or poor nutritional practices. It was noted that highly recommended food stuffs like beef, eggs and fish were avoided most by mothers during their pregnancies. This created the need for supplements for both children and their mothers especially during pregnancy and breast feeding periods. The solid food stuffs introduced sometimes become a challenge to the digestive systems of these young patients leading to chronic malabsorption, indigestion and diarrhoea.

About 73% of the patients were fed the recommended 3 times or more per day. Frequent feeding alone would not be sufficient if the quality and quantities taken were inadequate in terms of nutritional requirements, leading to the patients not meeting their recommended daily allowances.[27,51] The nutritional advisory committees from both Canada and the United States of America recommend risk groups to take at least 400 i.u of vitamin D per day for a non specified period and less than that would be considered

inadequate.[51,52] An alternative would be use of fortified proprietary milk but this may not be achievable due to the socio economic status of the patient's families in question.

Eighty one percent of patient's families food stuffs came from the market and most of the families were facing financial constrains. On the other hand most of the food would not be fresh and the hygiene sometimes questionable, since they were not obtained from farms directly and the places of sale are not highly inspected for hygiene and other standards. Some of these food stuffs may serve as sources of other microorganisms, hence worsening the health of the patients who already had other health challenges.

Little care regarding nutritional issues led to malnutrition and non exposure to ultraviolet rays since mothers and fathers were usually away during the day in search of a living and children were left with nobody mature at home to take care of them, as was also observed in a study carried out in Nigeria.[49]

Since these children could not take themselves into the sun or even feed themselves, the condition worsened. Hence prolongation of this situation led to rickets and other nutritional disease conditions.

4.2 Conclusion:

The risk of developing rickets among the patients in this study had a very strong relationship with economic status of their families. This was manifested by the type of food stuffs their families managed to access which was not a balanced diet.

The results support the hypothesis that deficiency or reduced availability of dietary calcium might have been of at least equal importance with vitamin D deficiency as a predisposing factor in the aetiology of rickets in the children and infants.

Although parents claimed to have knowledge of the disease, ignorance about the disease seemed to play a big role. Most parents brought their children to the hospital for other illnesses rather than rickets. A large number of the parents were relatively young and came from household / family settings which were young too. Early weaning to solid non nutritious food stuffs and short duration of exclusive breast feeding of children and infants seem to have had a major role to play in the aetiology of rickets in these young ones.

Inadequate sunbathing was another major factor, due to lack of enough space.

The first three born and ages of 3 months to 15 months carried a high risk of the disease.

Birth control or birth spacing of 2 years and above between siblings may reduce the risk of developing the disease but this study did not find enough children with poor spacing to address this question.

4.3 Recommendations:

i) All susceptible rickets patients of any age should be given standard doses of vitamin D, until proved not to be still under threat of the disease.

ii) Educative campaigns should be carried out nationwide regarding the predisposing factors of the disease, so as to prevent the disease from spreading.

iii) Further research studies regarding the relationship between rickets and socio demographic status of families and communities with low socio economic status in Kenya should done on a larger scale.

iv) The need to address the nutritional issues in low socioeconomic families is pre-eminent and should not be ignored at any cost by all stakeholders concerned.

v) Behaviour change in regard to the benefits of exposing children to sunlight is very important in preventing rickets and should be encouraged especially among the risk groups.

vi) Brochures with clear message regarding rickets should be prepared in various languages understandable to the populations and disseminated among the public

4.4 Study Limitations:

- Lack of enough resources to cover a large number of patients and which would have allowed involvement of other aspects.
- Limited time to carry out the study.
- Missing information from files and parents or guardians who did not respond to some questions

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Appendix ii: Work Plan

Activity	Execution Time
Proposal write up	Jan – March 2008
Submitting Proposal to the KNH Ethics & Research Committee	April 2008
Data collection	May – Aug 2008
Data Analysis	Aug -Sept 2008
Dissertation write up	Sept –Oct 2008
Defence of dissertation results	Nov 2008

Appendex iii: Budget for the Research Works

ITEM	DESCRIPTION	QTY	AMOUNT(KSHS)
Proposal development	Secretarial work, photocopying and binding. Literature review via internet 120hrs		60,000
Research Assistant	Staff Cost 1 research assistant in data collection for 3 months	1	45,000
External input-Statistician	Data Synthesis & Analysis Data entry final copies development	1	40,000
Contingencies; 15%			2,1750
Grand Total			**166,750**

TO BE FUNDED BY THE BELGIAN TECHNICAL COOPERATION RWANDA PROGRAM:

Appendix iv: Consent Form

I Dr. MWESIGYE John Patrick a final year master of pharmacy in clinical pharmacy, department of pharmaceutics and pharmacy practice, School of Pharmacy, University of Nairobi, am keen to request for a consent to carry out a research study on underlying causes of rickets in infants and children below five years of age admitted at Kenyatta National Hospital among which your child belongs.

The study will help medical practitioners to find other new dimensions in preventing and managing rickets and this may be a new base for a wider policy in the general management of this disease in our communities. I wish to inform you that this study involves no any examination either of the patient nor the parent or guardian; it also involves no any cost at all. This involves you, as apparent/guardian to furnish me with a range of information as is indicated in the questionnaire about the child and family.

To inform you also is that any information/data about the patient, parents or guardians and the family will remain confidential and will not be accessed by any unauthorized individuals or groups. Participation is voluntary and any refusal to take part will not in anyway interfere with your child's management, treatment and stay at KNH. You are free to ask any questions regarding this study and in case of any queries regarding this research study, may be forwarded to the Chairman KNH Ethics Committee, Professor Bhatt on Tel 020726300.

I....., hereby voluntarily consent to participate in the proposed research study on predisposing factors in rickets in infants and children.

The study has been explained by the investigator, I understand it involves no costs, examinations or laboratory tests.

I understand that my identity and information about the patient will be kept strictly confidential.

Signature of Parent/Guardian Signature of Investigator (Ph No 0203020064).

....

Date.. .../...../..... Date...../...../.....

Appendix v: Questionnaire Profoma

Personal History:

(A) Patient Study ID No..... Ward:.....

Sex: M ☐ F ☐ Home District.....

i Current Age (mo) :..... Current weight: ..... (g)

ii Birth History ☐ Term Preterm ☐

iii Birth weight: ..... (g)

iv Rank in family 1st ☐ 2nd ☐ 3rd ☐ Others specify .....

v Age gap between the patient and other siblings one before and one after the

Patient i.....mo, and ii-.....mo respectively.

vi Age at dentition i- ☐ 10 months and below ii- ☐ Above 10 months

x Age able to stand on self i- ☐ 18months and below ii- ☐ Above 18 months

(B)- Area of residence: .....

a- Cold ☐ b- Hot ☐ c- Cold and Wet ☐ d- Hot and wet ☐

i Size of compound ☐ Small ☐ Medium ☐ large

ii Is your child sun bathed at home ☐ Yes ☐ No

C- Home District.....

i- Mother Alive ☐ ☐ Deceased

ii- Father Alive ☐ ☐ Deceased

(D) Socio demographic Status

a- Gender of the household head M ☐ F ☐, Others specify.....

b-Parent / Guardian education level i) Non ☐ ii) Primary ☐ iii) Secondary ☐

iv) Tertiary ☐ v) Others .....

c- Guardian's or Parent's knowledge about the disease No ☐ Yes ☐

d- Parent's / Guardian's Occupation: .....

e-Who takes care of child at home.....

f- Parent's / Guardians Religion .....

(E) Nutritional History of the infant or child

i- Source of foods for the child and family

a- Market ☐ b- Home gardens/farm ☐ c- Both market and gardens/farm ☐

ii-Is child malnourished ☐ Yes ☐ No

iii- Is/was the child exclusively breastfed for 6 months Yes ☐ No ☐

a-Duration of Child's EBM in Months: .....,is child weaned Yes ☐ No ☐

iv- Complementary meals during breast feeding

a-.....

b- .....

v- Main weaning meals were(After stoppage of breast feeding):

a-.....

b-.....

c-.....

d-.....

vi - Frequency of feeding: Yes ☐ ≤ 2 times a day Yes ☐ ≥ 3 times a day

vii- At what age was the solid food introduced ..... mo

(F) Mothers Nutrition Patterns during Pregnancy and after delivery

i- Meals largely were:

a-During .....

b-.....

c- After.....

d- .....

ii- Food items craved for by the mother during pregnancy

a-.....

b- .....

iii-Food item avoided by the mother during pregnancy

a-.....

b-.....

(G) Medical History:

i- Diagnosis at admission

(a)..... (b).....

(c)..... (d) other.....

(ii) Past medical History

a- Any previous chronic illness to the child Yes ☐ No ☐

b-If yes above, which one(s)

c-.....

d-.....

(I) Drug History:

a- Long term use of drugs implicated Yes ☐ No ☐

b- If yes to the above, which one(s)

i-……

ii……

iii-……

(J) Any Chronic illness to the mother during pregnancy and after child birth. Yes ☐ No ☐

i-If yes to the above, which one(s)

(a) During……

(b) After……

(K) Mother's style of wearing clothes, especially covering of the head Yes ☐ No ☐

(M) Any family history of rickets Yes ☐ No ☐

(N) Are you going to clear your patient's medical bill? with easy ☐ difficulty ☐

Not able ☐

Appendix vi: Ethics and Research Committee approval

KENYATTA NATIONAL HOSPITAL
APPROVED
14 APR 2008
ETHICS & RESEARCH COMMITTEE

KENYATTA NATIONAL HOSPITAL
Hospital Rd along Ngong Rd
P.O. Box 20723, Nairobi
Tel: 2726300-9
Fax: 725272
Telegrams: MEDSUP", Nairobi
Email knhadmin@knh.or.ke

Ref: KNH-ERC/ 01/ 330

14th April, 2008

Dr. Nyiligira John
Dept. of Pharmaceutics and Pharmacy Practice
School of Pharmacy
UNIVERSITY OF NAIROBI

Dear Dr Nyiligira

RESEARCH PROPOSAL: "ASSESSMENT OF DISPENSING PRACTICES AND PATIENTS SATISFACTION WITH PHARMACEUTICAL SERVICES OFFERED AT KNH PHARMACIES" (P48/3/2008)

This is to inform you that the Kenyatta National Hospital Ethics and Research Committee has reviewed and **approved** your above cited research proposal for the period 14th April, 2008 – 13th April, 2009.

You will be required to request for a renewal of the approval if you intend to continue with the study beyond the deadline given. Clearance for export of biological specimen must also be obtained from KNH-ERC for each batch.

On behalf of the Committee, I wish you fruitful research and look forward to receiving a summary of the research findings upon completion of the study.

This information will form part of database that will be consulted in future when processing related research study so as to minimize chances of study duplication.

Yours sincerely

PROF A N GUANTAI
SECRETARY, KNH-ERC

c.c. Prof. K.M. Bhatt, Chairperson, KNH-ERC
The Deputy Director CS, KNH
The Dean, School of Pharmacy, UON
Supervisor: Dr. David Scott, Dept. of Pharmaceutics and Pharmacy Practice, UoN