THE RISK FACTORS ASSOCIATED WITH THE DEVELOPMENT OF NUTRITIONAL RICKETS IN CHILDREN AT KENYATTA NATIONAL HOSPITAL.

A DISSERTATION SUBMITTED IN PARTIAL FULFILLMENT

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DECLARATION

I hereby certify that this is my original work and that it has not been submitted in any other University.

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DEDICATION

TO MY HUSBAND AND DAUGHTER.

LIST OF ABBREVIATIONS

C.I.	Confidence interval
F	Female
I.U.	International Units
Kg	Kilograms
K.N.H	Kenyatta National Hospital.
M	Male
mmol/l	Millimoles/litre
n	Number
P.F.C.	Paediatric Filter Clinic
ug	Micrograms
umols/l	Micromoles/litre
U.O.N.	Universisty of Nairobi.

TABLE OF CONTENTS

LIST OF TABLES

ABSTRACT

A study to determine the risk factors associated with development of nutritional rickets in children at Kenyatta National Hospital was carried out . It was a case control study. Sixty three children under 18 months of age who had vitamin D deficiency rickets constituted the cases. A hundred and twenty six children marched with cases for age and sex and without rickets were recruited as controls. The children were recruited from the general paediatric wards and the paediatric outpatient clinic (POPC).

The age ranged from 2 1/2 months to 18 months with 9 months being the modal age. A higher percentage of children with rickets were born at a birth weight less than 2.5 kg
($P < 0.001$). Forty one percent of the rickets patients lived in flats. A higher percentage of children with rickets (51%) were not exposed to sunshine as compared to 3.2% of children without rickets ($P < 0.001$). Children with rickets were exposed to sunshine for fewer numbers of days per week and for shorter periods.

There was no difference in the availability of animal products in the diets of children with or without rickets. Cod liver oil was used equally by both groups of patients with or without rickets. However the cod liver oil was used as a therapy for coughs and colds and not a preventive measure against rickets.

In conclusion children with rickets were exposed to sunshine for fewer days per week, and for shorter duration. There was no significant difference in the availability of animal products in the diets of children in both groups.

It is recommended therefore that mothers should be educated on the prevention of rickets in their children through regular exposure to sunshine and appropriate use of vitamin D supplements.

INTRODUCTION

Rickets is a disorder in which mineralization of organic matrix of the skeleton is defective as a result of vitamin D deficiency. Defective mineralization occurs both in the bone and cartilaginous matrix of the growth plate. Bone remodeling and increase in thickness by periosteal bone apposition are active processes in growth which depend on deposition of calcium and organic phosphate. An alteration of the levels of these minerals whose physiological levels are maintained by vitamin D will result in osteomalacia and excessive accumulation of osteoid tissue at growing ~~tuds~~ ends of the skeleton. The skeleton will thus be affected in its two main functions as the mechanical support for other organs and the major mineral reservoir serving a large array of physiological functions[1,2,3].

Rickets results from a combination of various factors. Lack of exposure to sunlight has been found out as one of the predisposing factors to development of rickets. As early as 1882 Snaidecki observed that children living in Warsaw had a higher incidence of rickets compared to children living in rural areas[4]. At the beginning of the industrial revolution, people began to congregate into cities and their children played in crowded, sunless alleyways. The incidence of rickets increased dramatically during the industrial revolution especially in North Europe and North America. This observation prompted him to conclude that exposure to sunlight was the most important factor in the prevention and cure of rickets[5].

In Nigeria a study by Ekanem E.E. et al showed that children of elite and middle class parents developed rickets due to sunshine deprivation as they were kept indoors most of the day while their parents were away at work[6]. A study in Riyadh Saudi Arabia by Eldrissy A.T.H. showed that most rachitic children were living either in flats or traditional mud houses. The traditional mud house is high walled, with small high windows and like the flats, had no direct access to sunshine in contrast to rural houses that have courtyards where mothers and their children can

be exposed to sunshine in privacy. In Riyadh, and other tropical towns, sunshine is abundant but an "umbrella" is formed depriving the infants and their mothers access to direct sunshine[7]. Urbanization forms the main pillars on which this umbrella is pivoted, as it is associated with living in flats. These flats are devoid of direct sunshine, as ultraviolet light does not penetrate glass.

Poor vitamin D status in the mothers in pregnancy and after delivery predisposes the infants to development of rickets[8,9,10]. A study in Riyadh showed that mothers of rachitic babies were vitamin D deficient and paired maternal and cord blood vitamin D levels were low. In spite of this low vitamin D, calcium levels in cord blood were maintained within normal levels and were even higher than the corresponding maternal levels. These finding conform with the active transplacental transport of calcium hence the fetus maintains adequate bone mineralization and prevents congenital rickets. After birth, the infants are unable to maintain normal calcium levels due to the loss of active transplacental transport of calcium and lack of vitamin D reserve[11]. Beser et al showed that young mothers (below 18 years) and women who were not on contraception and hence not spacing their deliveries were more likely to have their children develop rickets[12]. Lulseged studying children with rickets in Ethiopia also showed that there was borderline significant association between the maternal age and development of rickets in their children[13].

Prolonged unsupplemented breast-feeding predisposes to development of rickets too[12,14,15]. Even with a relatively large vitamin D supply of 600 - 1000 I.U. per day, and adequate serum concentration of 1,25 dihydroxycholecalciferol in the mother, exclusively breastfed infants in countries like Norway and Finland rapidly deplete their vitamin D stores during winter when exposure to sunshine is negligible[14,16,17]. Daily exposure of the face and hands only for one to two hours is sufficient to maintain normal vitamin D levels in the body[7].

Strict vegetarian diets contribute to development of rickets in children. A study in Manitoba by Sanders A. showed that children on strict vegetarian diet were more at risk of developing rickets than those on both vegetarian and meat diets. Plant products have no or very little vitamin D. Fish is the richest source of vitamin D, having 200-750 micrograms/100g. Eggyolk has 3-10mcg/100g while cows milk has 0.02-0.1mcg/100g. As such a baby needs animal products in their diet and exposure to sunlight to get their daily requirement of vitamin D[12,16,18,19].

Prematurity is another important risk factor. Oyatsi D. in his study on rickets of prematurity at KNH found out that 58.8% preterms developed rickets[20]. Preterm babies are predisposed to development of rickets due to deficient stores of calcium and phosphate and also possibly vitamin D at birth. Apart from having a relatively small part of the skeleton which is calcified at birth preterm babies have rapid extrauterine growth which creates extra needs on minerals. In addition, renal functions in the preterm tend to be poor and vitamin D may not promote tubular reabsorption of phosphate. Because of their small size, preterm babies are less exposed to sunshine due to fear of hypothermia when they are taken outdoors[20,21,22].

In developed countries, the incidence of rickets dropped drastically after the introduction of vitamin D to evaporated milk (infant formulae), and with the introduction of health education talks on prevention of rickets in schools, maternity hospitals and also general practitioners clinics[23,24]. For instance the incidence of rickets dropped from 20% in 1938 to 2% in 1944 in a Massachusetts clinic[24,25]. Similarly the mean number of white children with florid rickets presenting each year at the Royal Hospital for sick children in Glasgow reduced from 21% between 1928 and 1955 to 4% between 1966 and 1971[26]. In Kenya, infant formulae fortified with vitamin D are available in the market but are expensive hence most families are not able to purchase them. The commonly used fresh milk e.g. from Kenya Co-op Creameries (KCC) is not fortified with vitamin D.

STUDY JUSTIFICATION

Rickets has been observed to be on the increase among inpatients in the paediatric ward at KNH[27]. In 1987, 0.037% of the total admissions in the general paediatric wards had rickets. This percentage has been rising over the years to 0.7% in 1994 and 1.4% in 1997[27]. Rickets is an important cause of morbidity and mortality. Children with rickets have been observed to have recurrent and/or non-resolving pneumonia, cor pulmonale among other complications[21]. This leads to prolonged or frequent hospital admissions. These complications can be avoided by prevention of rickets in children. The prevention of rickets through regular exposure to sunlight which is abundant in our environment, appropriate weaning foods and use of cod-liver oils are all relatively inexpensive. This study aimed at identifying some of the risk factors for developing rickets among children in Nairobi and hence recommend ways of preventing this easily preventable illness among children.

AIMS AND OBJECTIVES

AIM

To determine the risk factors associated with development of rickets in children aged 2 months to 18 months at Kenyatta National Hospital.

SPECIFIC OBJECTIVES

1. To determine the extent of sunshine deprivation on development of rickets in children aged 2 months to 18 months seen at Kenyatta National Hospital.

2. To determine the association between weaning foods and the development of rickets in children aged 2 months to 18 months seen at Kenyatta National Hospital.

METHODOLOGY AND STUDY DESIGN

This was a case control study.

Study area

The study was carried out at Kenyatta National Hospital general paediatric wards and paediatric out patient clinic. Kenyatta National Hospital is the national public hospital that serves as a referral and teaching hospital. It is also the main inpatient hospital for low and middle-income society in Nairobi and its environs. The paediatric outpatient clinic (POPC) which runs on Thursdays from 2 p.m. - 5 p.m. serves patients discharged from the wards and require follow-up and those from the paediatric filter clinic also requiring further investigation and follow-up. POPC sees an average of 400 patients per month. About 100 patients are seen each Thursday afternoon.

Sample Size

Sample size was determined by using EPI INFO 6. From lack of sunshine exposure as the measurable variable, the relative risk at 2 provided a case : control ratio of 1:2. Confidence interval was 95% and power 80%. A sample size of case : control of 63:126 was obtained.

Study population

Cases were children with rickets diagnosed clinically and radiologically. Controls were children of the same age and sex as cases but with no features of rickets clinically and radiologically and admitted to the ward for different ailments.

Inclusion Criteria

Cases: Children with confirmed rickets clinically and radiologically and normal renal function tests.

Controls: Two children with no rickets clinically and radiologically and

matched for a single case.

Children in both groups had to be accompanied by their mother and stay with the mother most of the day. Informed verbal consent was obtained from the mothers of the children before recruitment into the study.

Exclusion Criteria

Cases: Children with abnormal renal function tests.

Cases and Control: Children not accompanied by the mother.

Study Period

The study was carried out during the months of July and August 1999.

Study procedure

The investigator visited the general Paediatric wards on the post admission day and identified patients with rickets. Patients from POPC were recruited on Thursday afternoons in the clinic. A verbal consent was obtained from the mother. The mothers were interviewed and details recorded in the questionnaire (Appendix 1). A venous sample of blood was obtained from the patients and taken to renal unit laboratory for renal function tests. Patients found to have abnormal renal function tests were excluded from the study. Controls were children admitted on the same day as cases or attending POPC on the same day as cases.

The investigator checked the admission register after getting a case and the first two children in the register who satisfied the criteria for control were selected.. For POPC, two children seen after a case had been identified of same age and sex but had no rickets were recruited. All controls were sent for the x-ray of the left wrist. The x-rays confirming presence or absence of the rickets were reported by a radiologist.

Clinical signs of Rickets

Clinical rickets was diagnosed if two or more of the following signs were present; craniotabes, frontal bossing, bilateral widening of the wrist, rachitic rosary, Harrisons sulcus and pigeon chest[2,28].

Radiological signs of Rickets

Diagnosis of radiological rickets was made in the presence of any two of the following: generalized osteopenia, fraying and cupping of the distal ends of radius and ulna, and widening of the costochondral junction[29].

Renal function tests[30]

Normal creatinine levels are as follows;

Infant (upto 12 months) - 18 - 35 umol/l

Child - 27 - 62 umol/l

Normal Blood Urea Nitrogen (BUN);

Infant/Child - 1.8 - 6.4 mmol/l

DATA ANALYSIS

The data collected was fed into a computer and analysed using SPSSPC computer programme. To compare results for those with rickets and those without, the following tests of significance were applied; Chi-square (χ^2) test, Odds ratio and fisher's exact test.

ETHICAL CONSIDERATION

Permission to carry out the study was sought from the Kenyatta National Hospital Research and Ethical Committee. All mothers were advised on the benefit of exposing their children to sunshine regularly. They were also informed on diet that is rich in vitamin D. The children with rickets were put on appropriate treatment.

RESULTS

Data was collected over a two-month period (August and July 1999). A total of 189 patients were recruited with 63 being children with rickets (cases) diagnosed clinically and radiological. One hundred and twenty-six children without rickets (controls) were matched with cases for age and sex. The male to female ratio among patient with rickets was 1:1 (M=32, F=31). The age ranged from 2 1/2 months to 18 months with 9months being the modal age. Seventy-nine percent of the children with rickets were less than 12 months old.

Table 1: Age Distribution.

Age in months	Cases		Controls	
	N	%	N	%
Less than 6	16	(25.4)	32	(25.4)
6 - 12	34	(54.0)	68	(54.0)
Greater than 12	13	(20.6)	26	(20.6)

Of the patients with rickets that were recruited into the study, 25.4% were less than 6 months old, 54% were in age group 6 to 12 months old and 20.6% were older than 12 months. Overal, 79% of children with rickets were less than 12 months old.

Table 2: Gestation at Birth, and Birth Weight

Variable	Cases n %	Controls n %	P value	Odds ratio 95% CI
Gestation at birth				
Preterm*	9 (4.3)		0.022	
Term	54 (85.7)			
Birth weight (kg)**				
Less than 2.5	15 (24.2)		<0.001	4.43
Greater than or equal 2.5	47 (74.6)			1.6-12.3

* Preterm babies were those born at least 4 weeks before the expected date of delivery.

**One mother did not have the baby's clinic card and she could not remember the baby birth weight.

There was a high percentage (14.3) of babies born prematurely among patients with rickets as compared to 4.8 % for those without rickets (p value 0.022). This was statistically significant. Twenty four percent of the babies with rickets were born at a weight less than 2.5kg, whereas only 6.7% of the babies without rickets weighed less than 2.5kg at birth. Low birth weight increased the odds of rickets by a factor of 4.43.

Table 3: Type of Housing and comparison between different types of Houses versus occurrence of rickets.

Variable	Cases n %	Controls n %	P value	Odds ratio 95% CI
Type housing				
Flats	26 (41.3)	25 (19.8)		
*Plots	20 (31.7)	30 (23.8)		
**Slum/semipermanent	15 (23.8)	64 (50.8)		
***Single house	2 (3.2)	7 (5.6)		
Comparison between				
Flats	26	25	< 0.001	4.33
Slum/semipermanent	15	64		1.76-10.8
Flats	26	25	0.01	
Single house	2	7		
Flats	26	25	0.26	
Plots	20	30		

*Plot : Permanent house but with several units on one block.

**Slum/Semi-permanent: Several units on one building and constructed from mud, timber walls or paper.

***Single house: House with its own compound and includes farm house.

Patients with rickets living in Flats formed 41.3% of all cases (patients with rickets) as compared to 19.8% of those without rickets. Thirty two percent of babies with rickets lived in Plots while 23.8% of those without rickets lived in Plot type of housing. Fifty percent of children without rickets lived in slum/semi-permanent houses as compared to 23.8% of those with rickets. There were few children in the study who lived in single/farm houses. Three percent of the children with rickets lived in single houses compared to 5.6% of those without. Overal, there is a tendency towards higher occurance of rickets among children living in flats compared to other types of houses.

Table 4 : Exposure to sunshine, estimated duration and frequency of exposure to sunshine per week and age of starting to sunbathe.

Variable	Cases n %	Controls	P value	Odds ratio 95% CI
Exposure to sunshine				
Yes	31 (49.2)	122 (96.8)	<0.001	0.03
No	32 (50.8)	4 (3.2)		0.008-0.104
Estimated duration of exposure to sunshine per day				
Less than 30 mins	11 (35.5)	13 (10.7)	<0.001	
30-60 mins	20 (64.5)	58 (47.5)		
Greater than 60 mins	0 (0.0)	51 (41.8)		
Age of starting to Sunbathe				
Less than 1 month	9 (29.0)	61 (50.0)	0.035	0.41
Greater than 1 month	21 (71.0)	61 (50.0)		0.15-1.02
No. of days exposed to sunshine per week				
1-2	13 (41.9)	3 (2.6)		
3-4	8 (25.8)	18 (14.8)	0.045	
5-6	3 (9.7)	7 (5.6)		
7	7 (22.6)	94 (77.0)		
Preterm exposed to sunshine				
Yes	3 (33.3)	6 (100.0)	0.017	
No	6 (66.7)	0 (0.0)		

Children not exposed to sunshine were 99.97 times more likely to develop rickets than those exposed. Cases were exposed to sunshine for shorter periods (p <0.001) per day. Majority of them started sunbathing after the age of one month and they were exposed for fewer days per week than the controls (p value =0.01). All the babies without rickets who were born prematurely were exposed to sunshine as compared to only 33% of those with rickets. (Fishers exact test=0.017).

Table 5. Duration of exclusive breastfeeding, weaning foods and availability of animal products in the diet.

Variable	Cases		Controls		p value
	n	%	n	%	
Duration of exclusive Breastfeeding*					
Less than 3 months	27	(43.5)	65	(52.8)	
3-6 months	32	(51.6)	48	(39.0)	0.24
Greater than 6 months	3	(4.9)	10	(8.2)	
Weaning foods					
Porridge	23	(42.5)	54	(45.2)	
Mashed fruits	15	(25.4)	24	(45.2)	
Cow's milk	10	(15.8)	21	(16.7)	0.45
**Bananas/Carrots/Potatoes /Pumpkin/Spinach	14	(22.7)	25	(18.3)	
Cerelac/Weetabix	2	(3.2)	2	(3.2)	
Availability of animal products in diet					
Less than 3 months	27	(43.5)	65	(52.8)	0.5
3-6 months	32	(51.6)	48	(39.0)	
Greater than 6 months	3	(4.9)	10	(8.2)	

*Children still exclusively breastfeeding were not included.
**Bananas/Carrots/Potatoes/Pumpkin/Spinach: Various combinations were made from the above named foods and mashed together.

There was no significant difference in the duration of exclusive breastfeeding between children with rickets and those without rickets (p value =0.24). The types of weaning foods used by both patients with or without rickets were the same (p value=0.45). Porridge was used most as a weaning food (42.5% for children with rickets and 45.2% of children without rickets).

Available animal product in the diet of the patient was what was actually given to the patients. These included eggs, fish and beef. There was no significant difference in the eating of animal products by both cases and controls.

Table 6: Use of vitamin D supplement

Variable	Cases n %	Controls n %	P value	Odds ratio 95% CI
Use of vit.D supplement				
Yes	41 (66.8)	50 (31.6)	0.002	2.6
No	23 (33.2)	76 (68.4)		1.35-5.19
Type of vit. D supplement				
Scotts emulsion	36 (87.8)	45 (90.0)	0.84	
Seven seas	5 (12.2)	5 (10.0)		
Appropriate dose of Vitamin D Supplement				
Yes	11 (26.8)	19 (38)	0.25	
No	30 (73.2)	31 (62)		
Duration of use of vit. D Supplement				
Less than 1 month	27 (67.5)	32 (64.0)		
1-2 months	8 (20.0)	10 (20.0)	0.8	
3-4 months	5 (12.5)	16 (32.0)		

Sixty seven percent of children with rickets were already taking some form of vitamin D supplement as compared to 32 % of children without rickets. This was statistically significant with a P value of 0.002. Scotts emulsion was used by majority of the children on supplement among both cases (88%) and controls (90%) Majority of children with or without rickets had used vitamin D supplement intermittently. Of children who used vitamin D Supplement, 73.2% of children with rickets and 62% without rickets used doses below the recommended dose by the manufacturers.

DISCUSSION

The study aimed at determining the factors associated with development of nutritional rickets in children at Kenyatta National Hospital. The male to female ratio was 1:1. Previous studies have shown male to female ratio to be 1:1 among patients with rickets[6,12]. However, a study in Ethiopia showed a male preponderance[13]. Seventy nine percent of patients were less than 12 months old. This was again in keeping with earlier studies where the modal age for rickets was 6 to 12 months[12, 13, 31]. Our modal age was 9 months.

There was a statistically significant difference in the gestational age at birth and occurrence of rickets with 14.3% of the children with rickets having been born prematurely ($\leq$ 36/40). Prematurity is a known predisposing factor to occurrence of rickets[20,22]. Only 5% of babies without rickets were born prematurely. Oyatsi in his study showed that 58.8% of premature babies born at Kenyatta National Hospital developed rickets[20]. Twenty four percent of children with rickets and 6.7% of those without rickets had a birth weight less than 2.5 Kg. The small for gestational age and premature babies have rapid growth postnatally hence are at risk of developing rickets. In addition, preterm babies are born with inadequate stores of calcium and phosphorus which normally accumulate in the third trimester. Postnatal feeding does not allow for adequate supply of calcium and phosphate as at levels in utero due to inefficient intestinal absorption. The preterm infants are also considered to be frail hence they are kept indoors most of the time. When they are taken out of the house, they are usually wrapped in several layers of clothing to prevent hypothermia and in turn are not exposed to adequate sunshine[20,32, 33].

Rickets was more common in children living in flats than other types of housing. Of the children who lived in flats, 51% of them developed rickets. This formed 41.3% of all the children with rickets. Staying in flats increased the likelihood

(odds) of developing rickets by 4.43. A study in Riyadh Saudi Arabia also showed that children living in flats were more at risk of developing rickets than those in other types of houses. The reason for children living in flats developing rickets was attributed to lack of exposure to sunshine[7]. The children are not taken out to sunbathe as there is limited privacy for the children and their mothers. Ultraviolet light at wavelength 296 to 310 nm required for vitamin D synthesis does not penetrate through glass hence a child who stays indoors most of the time is devoid of this light.

In slum/semi-permanent type of housing, the children and their mothers spend a significant period outside the house. The houses in slum areas are usually small, have no water or toilet facility inside hence the mothers have to go outside to wash or use the communal toilets. In the process of doing their day to day work the babies get exposed to sunshine.

All the babies born prematurely but exposed to sunshine (in the control group) did not develop rickets. Only 33.3% of babies with rickets born prematurely were exposed to sunshine. As such preterm exposed to adequate sunshine will not develop rickets. Children with rickets were exposed to sunshine for fewer days per week compared to children without rickets. Controls were exposed to sunshine for an average of 6 days per week while cases had an average of 2 days per week. A baby needs on average 1 to 2 hours of daily sunshine exposure to prevent rickets.

There was no significant difference in the duration of exclusive breastfeeding between the two groups. Ninety percent of babies in both groups had been weaned by the age of six months. Forty-five percent of babies without rickets and 42% of those with rickets were weaned on porridge. Porridge was mainly made from millet flour and enriched with milk. Mashed fruits, cow's milk were the other foods commonly used for weaning of babies. There was no statistically significant difference in the types of foods used for weaning the cases and controls. All the

above mentioned foods are poor in vitamin D content, hence the need to supplement the preterm babies with vitamin D and to adequately expose the children generally to sunshine.

There was no significant difference in the availability of animal products in the diets of children with or without rickets. A common finding was that majority of the mothers gave their children soup only when they had cooked meat or fish for the family. Sanders showed that children who were on vegetarian diet were more likely to develop rickets than those on a mixed diet[19]. A few mothers enriched the porridge by adding an egg to the porridge or mixing the maize or millet with fish (omena) before grinding. It was not possible to quantify the amount of animal products (except eggs) given to the babies as most mothers talked of giving a piece or two. The size of the piece of meat/fish definitely varies depending on the cutter. Accessbility to sunlight could have been the all important factor that made the difference in the occurrence of rickets.

A higher percentage of patients with rickets (62%) as compared to 32% without rickets had used cod liver oil. The codliver oil was not prescribed by a medical person, hence the dosage used by both groups was below the recommended dose by manufacturer for the prevention of rickets. Eighty eight percent of the patients with rickets and 77% of those without rickets used codliver oil doses below the recommended dose. Secondly, cod liver oil was used intermittently and for short periods as a treatment for coughs and colds and not as prophylaxis against rickets. As children with rickets are more prone to developing acute respiratory tract infections, it is therefore not surprising that a higher percentage of children with rickets used cod liver[35] oil more than those without. None of the babies born prematurely in the control group were on Vitamin D supplements. Sixty-six percent of the babies with rickets born prematurely had used vitamin D supplement. However, it was for short duration ranging from two weeeks to one month.

Multivitamins are another source of vitamin D supplement in our market. However, not all the brands in the market contain vitamin D. A survey of different multivitamins syrups at a private pharmacy showed that 40% of the multivitamin did not contain vitamin D (Hematinics were not included). The multivitamin syrups containing vitamin D had different strenghts e.g. mixavit had 400 i.u per 0.6mls, Kiddi pharmaton had 400 i.u per 5ml, Rinnavit had 200 i.u per 5 mls. Mothers were not able to say precisely which brand of multivitamin had been used.

Overall, this study has shown that sunshine deprivation is an important cause of vitamin D deficieny rickets in children seen at Kenyatta National Hospital.

CONCLUSION

1. Children with rickets were exposed to sunshine less frequently and for shorter periods than those without rickets.

2. There was no significant difference in the availability of animal products in in the diets of children with or without rickets.

RECOMMENDATION

There is need to educate mothers on prevention of rickets in their children through exposure to sunshine and adequate supplementation of vitamin D.

APPENDIX I

QUESTIONNAIRE

1. Study No: _____ case / control
2. Name _____
3. Sex
   1. Male
   2. Female
4. Age in months _____
5. Weight _____kg
6. Gestation at Birth _____weeks
7. Birthweight _____ kg
8. Duration of stay in Nairobi(months)_____
9. Area of residence? _____
10. Type of House
    1. Flat
    2. Temporary/Semi permanent/slum
    3. Single house
    4. Others
11. Who stays with the child most of the time.
    1. Mother
    2. House help
    3. Others (specify)
12. Is the baby taken out to sunbathe regularly

    0— No

    1— yes
13. If yes to Question 12, how many days in a week? _____
14. For how long does the baby stay in the sun? _____
15. At what age did you start taking the baby out to sunbathe? _____

16. Which parts of the body are exposed while he/she is in the sun?

_____

17. Did the baby breastfed at all?

0. No

1. Yes

18. Duration of breastfeeding? _____

19. At what age was the baby weaned? _____

20. What foods was the baby weaned on? _____

21. What is the baby's current diet? _____

_____

22 What meat products (including eggs) does the baby eat?

_____

23. How often does the baby eat above meat products in a week?

_____

24. Has the baby ever taken any of the following medicine?

- Scotts emulsion
- Seven seas
- Haliborange
- Multivitamin

1. Yes
2. No

25. If yes to Question 24 state dose and duration of use of each of the drugs

Name	Dose	Duration
1._____	_____	_____
2._____	_____	_____
3._____	_____	_____
4._____	_____	_____

APPENDIX II

Content of vitamin D in different preparations of codliver oil[36].

1. Scotts emulsion

   Vitamin D content: 85I.U./15ml

   Recommended dose 0 - 1year 5ml per day.

   1 - 6 years 15ml per day

2. Seven Seas

   Vitamin D content: 800I.U./10ml

   Recommended dose 0 - 6 months 2.5ml per day.

   7 - 12 months 5ml per day.

   > 1year 10ml per day.

Source: Packets of respective preparation.

APPENDIX III

VITAMIN D CONTENT OF SOME COMMON FOODS[37]

TYPE OF FOOD	ug cholecalciferol/100g
1. Fish	200 - 750
2 Egg yolk	3 - 10.
3. Butter	2 - 9
4. Egg	1 - 1.5.
5. Cows milk	0.02 - 0.1
6 Human milk	0.05

Recommended daily intake for children(0 - 6 years): 10ug

100 I.U.is equivalent to 2.5 ug.

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