STUDY ON PREVALENCE OF HYPOVITAMINOSIS D IN EXCLUSIVELY BREASTFED BABIES IN AN URBAN REFERRAL CENTRE

Dissertation Submitted in fulfilment of requirements for the degree of

M.D. Paediatrics

BRANCH VII

THE TAMILNADU Dr. M.G.R MEDICAL UNIVERSITY

CHENNAI

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INSTITUTE OF CHILD HEALTH AND HOSPITAL FOR CHILDREN

EGMORE, CHENNAI.

CERTIFICATE

This is to certify that the dissertation titled, **"Study on prevalence of Hypovitaminosis D in exclusive breastfed babies"** submitted by **Dr.V.Prabu,** to the Faculty of Pediatrics, The Tamilnadu Dr.M.G.R Medical University, Chennai, in partial fulfillment of the requirements for the award of M.D. Degree (Pediatrics) is a bonafide research work carried out by him under our direct supervision and guidance, during the academic year 2010-2012.

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DECLARATION

I, **Dr. V.Prabu**, solemnly declare that the dissertation titled **"Study on Prevalence of Hypovitaminosis D in exclusive breastfed babies"** has been prepared by me.

This is submitted to the Tamilnadu Dr. M.G.R. Medical University, Chennai in partial fulfillment of the rules and regulations for the M.D. Degree Examination in Pediatrics.

Place : Chennai

Date : **Dr. V. PRABU**

SPECIAL ACKNOWLEDGEMENT

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I sincerely thank all the children and their parents who have submitted themselves for this study.

INSTITUTIONAL ETHICS COMMITTEE
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CERTIFICATE OF APPROVAL

To
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Dear Dr. V. Prabu

The Institutional Ethics Committee of Madras Medical College reviewed and discussed your application for approval of the proposal entitled "Prevalence & Risk factors for hypovitaminosis D in exclusively breast fed babies" No. 20072011.

The following members of Ethics Committee were present in the meeting held on 21.07.2011 conducted at Madras Medical College, Chennai -3.

1.	Prof. S.K. Rajan, MD	-- Chairperson
2.	Prof. V. Kanagasabai, MD Dean, Madras Medical College, Chennai-3,	-- Deputy Chairman
3.	Prof. A. Sundaram, MD Vice Principal , Madras Medical College, Chennai -3	-- Member Secretary
4.	Prof R. Sathianathan , MD	-- Member
5.	Prof R. Nandhini, MD Director, Institute of Pharmacology, MMC, Ch-3	-- Member
6.	Prof. Geetha Subramanian MD. DM Prof & Head , Dept. of Cardiology, MMC, Ch-3	-- Member
7.	Prof. Pregna B. Dolia, MD Director , Institute of Biochemistry, MMC, Ch-3	-- Member
8.	Prof. C. Rajendiran, MD Director , Institute of Internal Medicine, MMC, Ch-3	-- Member
9.	Thiru. A. Ulaganathan Administrative Officer, MMC, Chennai -3	-- Layperson
10.	Thiru. S. Govindasamy . BA.BL	-- Lawyer
11.	Tmt. Arnold Soulina MA	-- Social Scientist

We approve the proposal to be conducted in its presented form

Sd /. Chairman & Other Members

The Institutional Ethics Committee expects to be informed about the progress of the study, any SAE occurring in the course of the study, any changes in the protocol and patient information / informed consent and asks to be provided a copy of the final report

Member Secretary, Ethics Committee

CONTENTS

INTRODUCTION

Exclusive breast-feeding is recommended up to 6 months of age with all its beneficial effects on child survival[1]. Globally as many as 1.45 million lives are lost due to suboptimal breast-feeding in developing countries. WHO analysis of childhood deaths has listed suboptimal breast-feeding as one of the most powerful shared risk factors and estimated that 1.3 million deaths can be prevented in 42 high mortality countries by increasing the level of breast-feeding amongst infants.The increase in the practice of breast feeding associated with the belief that "breast is best" and that breast milk does not require supplementation because it is a babys "perfect food" may lead to decreased 25-hydroxy vitamin D (25-OHD) intake from other sources and thereby causing rickets[1]. The prevalence of hypovitaminosis D in exclusively breast fed infants to be 82 ,52 and 20 % from UAE[5],Pakistan[6] and China[7] respectively but there is a paucity of data from India regarding the same.

The newborn infant born to a vitamin D replete mother is protected from vitamin D deficiency for the first few months of life as 25-OHD crosses the placenta readily and neonatal levels approximate two thirds of maternal serum concentrations[1]. Serum 25-OH D has a

half-life of approximately 3 wk, thus providing some protection against vitamin D deficiency for a couple of months even if the young infant does not receive vitamin D whereas in an infant who is exclusively breast-fed and who gets minimal sunlight exposure or an infant who is on a non fortified milk substitute the risk of developing vitamin D deficiency rickets by 4-6 months of age is very high[1].

It has been estimated that breast milk from a vitamin D replete mother contains between 20 and 60 IU/l of vitamin D[18] and hence adequate intake of vitamin D cannot be met with human milk as the sole source of vitamin D in a breast-feeding infant [18,19].

Hence a study was undertaken to identify the prevalence of subclinical hypovitaminosis D in exclusive breast fed babies in tertiary care hospital.

REVIEW OF LITERATURE

METABOLISM AND FUNCTIONS OF VITAMIN D

Most vertebrates ,including humans,obtain most of their vitamin D requirement from exposure to sunlight.During sunlight exposure,the precursor of cholesterol,7-dehydrocholesterol,absorbs ultraviolet B radiation (290-315mm) whih results in bond cleavage to form previtamin D3[(3)].Previtamin D3 ,which is sandwiched in between the lipid bilayer of the plasma membrane,undergoes rapid transformation to vitamin D3[(3)].

Both vitamin D2, which comes from yeast and plants and vitamin D3, are metabolized in a similar fashion in the liver and kidney. 25 hydroxylation occurs in the liver and 1 alpha-hydroxylase in the kidneys convert it into active 1, 25(OH)2 Vitamin D 3[(3)].Vitamin D plays a critically important role, not only calcium metabolism, but has similar functions as calcium in regulating cell proliferation, differentiation, and other metabolic roles[(3)].

EXPOSURE TO SUNLIGHT AND VITAMIN D:

Since most of the vitamin D requirement for children and adults is met from sunlight exposure,any alteration in the penetration of ultraviolet B radiation to the skin can dramatically affect the cutaneous production of vitamin D3.The natural sunscreen melanin in the skin markedly reduces the cutaneous production of vitamin D3.Thus ,the increased skin melanin pigment and limited sun exposure explain the high incidence of vitamin D deficiency in African-American breast fed infants and adults[(9)].

Vitamin D deficiency in infants and young children results in secondary hyperparathyroidism,which helps maintain serum calcium in normal range.However, because of phosphaturic effect of PTH,serum phosphorus levels decline.The inadequate calcium x phosphate product results in poor skeleton minerlization.There is a disruption in chondrocyte maturation and inhibition of the growth plate mineralization.This results in a widening of the epiphyseal plates at the end of the long bones in the arms and legs.When rachitic children begin to stand, gravity causes either an inward or outward bowing or concavity of the long bones in the lower extremities,resulting in either knock knees or bow legs[(4)].

BIOLOGICAL ROLE OF VITAMIN D

It is now recognised that vitamin D is not only important for absorption of calcium and maintaining a healthy skeleton but also a wide range of other biologic functions. As does calcium ,1,25(OH)2 D regulates cellular growth and induces maturation[4].Vitamin D receptors exist in a wide variety of tissues cells throughout the body that include the brain, breast, colon, prostate, skin, gonads and activated T and B- lymphocytes.It appears that 1, 25(OH)2 D has subtle but important biologic function in all these tissues and cells.In addition to these cells having a vitamin D receptor, it is now recognized that they also have the enzymatic machinery to produce 1,25 (OH)2 [4]. Thus vitamin D may be important at a more fundamental level where it is activated locally in many cells and tissues in the body to regulate cell growth regularly. Activated T and B –lymphocytes have receptors for 1,25(OH)2 Vitamin D[4].

DIETARY SOURCES

Very few foods contain preformed vitamin D, which is why children as well as adults are at risk for vitamin D deficiency.The flesh of oily fish ,such as salmon and mackerel,contain variable amounts of vitamin D3[(4)].Thus , eating salmon or mackerel or other oily fish 3-4 times a week is usually adequate to provide ones vitamin D requirement.Cod liver oil and oils from other fish such as sharks also are excellent sources of vitamin D .Very few foods are fortified with vitamin D[(4)].

CALCIUM METABOLISM

Calcium is critical for a many biological processes in the body.Ninety percent of the calcium in humans is present in the skeleton,while the rest is present in the extra cellular fluid and soft tissues..Skeletal calcium maintains skeletal integrity,and also provides a small(1%) exchangeable component,which plays a crucial role in extra cellular calcium homeostasis[(2)].

CALCIUM ABSORPTION:

About 30% of dietary calcium is absorbed both by an active trancellular process(chiefly in the duodenum), and a passive paracellular process(throughout the small intestine)[4].The trancellular mode of transport is vitamin D dependant and predominates if the calcium intake is below 400 mg/day[4].When calcium intake is low,there is stimulation of parathyroid hormone secretion, which results in increased Calcitriol synthesis. This increases fractional calcium absorption. Luminal factors like phosphates, oxalates, phytates and fibre can reduce calcium absorption[2].

ROLE OF VITAMIN D AND PARATHORMONE:

Extracellular ionized calcium concentration is tightly maintained within a narrow range in humans. Parathyroid hormone and vitamin D are the most important regulators of extracellular calcium concentration[2].The parathyroid cell is exquisitely sensitive to minor alteration in serum calcium level, and senses calcium by a cell surface G protein linked, calcium sensing receptor[4].Lowering of serum

calcium results in a rise in PTH[4].This rise in PTH normalizes the reduced serum calcium level because of its action on the kidney (by increasing calcium reabsorption), bone (by increasing calcium efflux from bone), and intestine (by increasing calcium absorption,indirectly by its effect on calcitriol production).Long term hypocalcemia,as occurs in renal failure,results in hyperplasia of the parathyroid glands and marked increase in PTH secretion[4].

DIETARY SOURCES:

The recommended daily dietary intake of elemental calcium is 400-600 mg for infants,800 mg for children and 1200mg for adolescents.The best sources are milk and milk products,certain fish,and to a lesser extent green leafy vegetables and lentils[4].

PHOSPHORUS METABOLISM

About 85 % of body phosphorus is in the skeleton while the rest is in the extracellular fluid and soft tissues.A nonsaturable diffusion process via intercellular route absorbs about 65% of dietary phosphorus .Cellular transport via a vitamin D dependant active

sodium phosphorus co-transport mechanism accounts for the remainder.The jejunum is the main site for phosphorus absorption Serum phosphorus concentration is maintained by a variety of homeostatic mechanisms, but control is not as tight as for calcium, and the serum level fluctuates over a fairly wide range[4].Although phosphorus deficiency may lead to increase in 1,25(OH)2 D3 synthesis in the kidney (which enhances intestinal absorption of phosphorus), and an increased efflux of phosphorus from bones ,these are not the major factors controlling phosphorus homeostasis.The regulation of phosphorus concentration is principally achieved by varying the proximal tubular reabsorption. Deficiency of phosphorus results in reduced renal phosphate excretion[4].

The daily requirements of phosphorus in infancy, childhood and adolescence are similar to those for calcium. Phosphorus is found in most commonly consumed foods, including milk, whole grain cereals and legumes[4].

DISORDERS OF VITAMIN D METABOLISM

Calcitriol (1,25 dihydroxy vitamin D3),the active metabolite of vitamin D,promotes the intestinal absorption of calcium,while the secondary actions include the enhancement of bone resorption.Owing to these effects,which raise the serum calcium level,any deficieny or resistance to Calcitriol leads to hypocalcemia[(2)].

RICKETS

Rickets is a childhood disorder in which there is a lag in the rate of mineralization of the cartilage matrix at the growth plate .Osteomalacia refers to the failure to mineralize osteoid or bone matrix throughout the skeleton and occurs in the adults.The resulting soft bones can bend resulting in deformities and also fractures[(4).]

RICKETS can be caused by deficiency of vitamin D ,calcium, or phosphorus. It is also caused by a number of distinct abnormalities in metabolism of vitamin D [(3).]

VITAMIN D DEFICIENCY:

Vitamin D deficiency is usually due to inadequate intake or inadequate exposure to sunlight. It can also be induced by anticonvulsant therapy and malabsorption[4].

Nutritional rickets:

Francis Glisson from England published the first detailed description of rickets in 1650[4].The industrial revolution of the 19th century resulted in a marked increase in the incidence of rickets ,as the smoke emitted from factories prevented UV rays from reaching the earth's surface.The early part of the 20th century saw the recognition of vitamin D deficiency has become a rarity in most of the developed world due to fortification of foods with vitamin D ,nutritional rickets is still common in developing countries[4].

The following are the studies conducted in different countries regarding thr prevalence of Vitamin D deficiency

- Ovesev L et al reported increase incidence of vitamin D deficiency on geographical differences with particular references to European countries[8].

- Gannage-Yared MH et al reported similar observations in the prevalence of Hypovitaminosis D in Europe ,Africa and the Middle east countries[10].
- Hashemipour S et al reported a high prevalence of vitamin D deficiency in Tehran[11].
- Holvik K et al reported high incidence of Vitamin D deficiency in immigrants in Oslo Norway[13].
- Robinson BD et al reported increased prevalence and reemerging burden of rickets in Sydney ,Australia[12].
- Pal BR et al reported regarding the resurgence of rickets in the United Kngdom[15].
- Shaw NJ et al reported high prevalence of vitamin D deficiency in UK Asian families[14].
- Gessner BD et al reported the increasing incidence of nutritional rickets among breastfed black and Alaska native children and also recommended vitamin D supplementation[16].

Low levels of vitamin D were noticed even in healthy individuals in various reports.

- Gessner BD reported low levels of 25-hydroxy vitamin D levels among healthy children in Alaska[17].
- In India Goswami R et al reported the prevalence and significance of low 25-hydroxy vitamin D concentrations in healthy subjects in Delhi[20].
- Gordon CM et al reported the incidence of vitamin D deficiency in healthy adolescents wherein the prevalence was highest in Africa American teenagers than US adolescents[18].
- El-Hajj Fuleihan G et al reported the prevalence of hypovitaminosis D in healthy school children especially in girls in Lebanon[19].

AGE GROUP AT RISK FOR VITAMIN D DEFICIENCY

Exclusive breast fed infants and adolescents are the two pediatric age groups particularly at risk for vitamin D deficiency.

Contributory factors in infancy for vitamin D deficiency are

A) Poor maternal vitamin D reserves
B) Inadequate sunlight exposure.
C) Low levels of vitamin D in breast milk

The following are reports regarding the vitamin D deficiency in infancy with particular reference to breastfed infants.

- Atiq M et al reported vitamin D deficiency in breastfed infants in Pakistan[6].
- Dawodu A et al concluded that low vitamin D intake in exclusively breastfed babies was contributed probably to low maternal vitamin D status and recommended vitamin D supplementation for both the mother and baby[5].
- Bhalala V et al reported that subnormal maternal vitamin D status is associated with vitamin D deficiency in newborns and persists in exclusively breast fed infants[21].
- Iqbal SJ et al reported the continuing problem of vitamin D deficiency in pregnant Asian women and their offspring[24].
- SD Shenoy et al reported that maternal vitamin D deficiency led to refractory neonatal hypocalcemia and the development of nutritional rickets in their infants[25].
- Kreiter SR et al reported the prevalence of nutritional rickets in African ,American breastfed infants and recommended that all dark skinned breastfed infants and children should receive vitamin D supplementation[22].

- Specker BL et al reported that adequate exposure to sunshine,reduces the incidence of vitamin D deficiency in nursing mothers and the exclusively breastfed infants[23].

- Lyn et al also reported increased prevalence of nutritional rickets in breastfed infants in USA,who presented with hypocalcemic seizures during the first year of life and recommended that all breastfed infants need vitamin D supplementation at an early age and continued throughout the duration of breastfeeding[26].

- Ahmed I et al reported similar incidence of hypocalcemic seizures due to vitamin D deficiency in breastfed infants in Pakistan[27].

- Hatun et al reported increased subclinical Vitamin D deficiency in adolescent girls who wear concealing clothing and recommended vitamin D supplementation in the adolescent girls[28].

- SBS et al reported that Vitamin D deficiency is observed in exclusive breastfed infants and can present with hypocalcemic seizures.[1]

- Price DI et al reported a breastfed infant with clinical evidence of rickets and with dilated cardiomyopathy who responded well to calcium and vitamin D supplementation[29].

- Gulati S et al wherein children presented with congestive cardiac failure due to hypocalcemic cardiomyopathy due to vitamin D deficiency[30].

- Daaboul et al reported the incidence of symptomatic vitamin D deficiency in infants secondary to maternal vitamin D deficiency in USA and recommended vitamin D supplementation to the pregnant , breastfeeding mothers and their infants[31] .

- Greer Marshal et al reported that vitamin D unsupplemented ,breastfed infants had no evidence of vitamin D deficiency during the first 6 months of life.However Gessner BD et al concluded that exclusive breastfeeding is a riskfactor for vitamin D deficiency in early infancy[32].

- Reports showing increasing rates of rickets due to insufficient exposure to sunlight and inadequate Vitamin D intake are the cause for serious concern as concluded by Molgaard et al[33].

Clinical features of rickets :

Classic clinical findings of rickets in infants include

-Frontal bossing

-Widened fontanelle and sutures

-Softening of the occipital bone on thumb pressure in infants(Craniotabes)

-Enlarged costochondral junctions.

-Harrison's sulcus and other chest deformities.

-Widening of wrists and ankles is commonly seen.

-Genu varum(bow legs) is common in toddlers.

-Genu valgum (knock knees) is common in older children

-Undermineralised bone is soft and more likely to bend than break , but fractures of long bone may occur.

-A potbelly may be seen due to weakness of the abdominal muscles.

-Lower respiratory tract infections are common in infants.

-proximal muscle weaknes leads to the typical waddling gait[(2)].

In severe vitamin D deficiency there may be anemia, thrombocytopenia, bone marrow hypercellularity and extramedullary hematopoiesis.

Seizures may occur due to hypocalcemia, especially in infants and adolescents[(34)]. Bone pain can occur[(4)]. Development of clinical vitamin D deficiency rickets is dependant not only on severity of vitamin D deficiency but also on duration of deficiency.

Stages of clinical vitamin D deficiency:

Stage 1: hypocalcemic symptoms

Stage 2: skeletal deformities

Stage 3: worsening of symptoms of 1&2

Bio-chemical Findings:

Stage 1: 25(OH)D ↓, ↓Ca, normal phosphate &1,25(OH)D may ↑or normal, serum PTH ↑.

Stage 2: 25(OH)D further ↓,normal calcium ,↓phosphate. & slight ↑in alkaline phosphatase.

Stage 3: Severe ↓ in 25(OH) D, ↓ ca, ↓ phosphate & ↑ alkaline phosphatase.

Radiological findings:

Classic radiological findings are seen in the epiphyseal ends of long bones.Due to accumulation of uncalcified cartilage,the metaphysis is widened and cupped and may appear distorted or frayed[4].In the very young children,symptomatic hypocalcemia

manifests even before any radiological changes occur due to vitamin D deficiency.Features of more advanced disease include delayed appearance of ossification centres and cortical thinning, bowing of lower limb long bones and fractures.

Therapy for Nutritional rickets:

In the management of Nutritional Rickets ,a single dose of Inj.Arachitol 6 lakhs units intramuscular,followed by good intake of elemental calcium(800mg/day in children)[(4)].Healing with normalization of serum calcium and phosphrous takes 1-4 weeks and serum alkaline phosphatase takes place in 6-12 weeks.Radiologically. healing is first evidenced by the appearance of linear deposits at cartilage/bone apposition in long bones. Bowing of long bones may disappear within few years/ even as long as 4-5 years.

To prevent nutritional rickets,daily dietary intake of calcium 50-100 mg/kg/day coupled with intake of vitamin D 400 IU daily on adequate exposure to sunlight to the child[(4)].

- The AAP pediatric nutrition handbook suggests that dark skinned breastfed infants with inadequate exposure to sunlight need supplementation at 400 IU/day[35]
- Lawrence et al recommended that all infants , including those who are exclusively breastfed, have a minimum intake of 200 IU of vitamin D per day beginning during the first 2 months of life and also to be continued throughout childhood.[36]

Recommendations for particularly pregnant and nursing mothers tend to prevent rickets in infancy. In the presence of anticonvulsant therapy, rickets can be prevented by a minimally high intake of vitamin D (800 IU).Treatment and prevention of rickets in malabsorption may need high doses (10,000 IU daily.).Alternatively 1,00,000 units can be given once in a few weeks to mothers[4].

- Gunsul Kutluk et al reported the increasing incidence of nutritional rickets in Turkey in the age group less than 2 years due to intake of cows milk without any supplementation of vitamin D associated with inadequate exposure to sunlight and recommended a combination of oral calcium with vitamin D to prevent the morbidity due to nutritional rickets as recommended by Pettifer et al in their study[40].

- In the UK the Department of Health recommends 400 IU for the pregnant and lactating women,340 units for infants under 6 months and 280 units for children under 4 years witn no recommendation for older children[41].
- There is a need for global perspective for the current status of Vitamin D deficiency in exclusive breast fed infants and older children worldwide and the need for fortification of policies regarding vitamin D intake as recommended by Calvo et al[42].
- Vitamin D deficiency secondary to inappropriate feeding has been reported by Huang et al[43]. There are also recommendations for resetting RDA for vitamin D intake as emphasized by Whiting et al[44].
- Also Vitamin D deficiency is caused by imoroper lifestyle in children which could be best managed by parental education ,dietary modification,proper weaning and increase in exposure to sunlight as recommended by Mikayo et al[45].

STUDY JUSTIFICATION

- ✓ Vitamin D deficiency and rickets more than 6 months is well known entity.But ,vitamin D deficiency in exclusive breastfed babies < 6 months not well known.
- ✓ Most of the studies from Pakistan ,UAE & China.Indian studies are lacking.No studies from our institution.
- ✓ A detailed study may throw light on this subject and will be useful in early diagnosis of vitamin D deficiency and appropriate measures to prevent the same

AIM OF THE STUDY

To determine the prevalence of subclinical hypovitaminosis D among exclusively breast fed babies.

SUBJECTS AND METHODS

STUDY DESIGN : Prospective Descriptive study

PLACE OF THE STUDY : INSTITUTE OF CHILD HEALTH & HOSPITAL FOR CHILDREN

PERIOD OF THE STUDY : NOV 2010 TO DEC 2011

STUDY POPULATION :

- All exclusively breast fed healthy term babies with birth weight > 2.5 kg
- Babies not on multivitamin drops

SAMPLE SIZE : 40 Babies

Ethical committee clearance was obtained from the Institutional Review Board

METHODOLOGY

This was a descriptive study conducted in a tertiary care exclusive pediatric hospital in Chennai.All term healthy babies on exclusive breast feeding,who were attending the well baby clinic of IOG and VPD clinic of ICH & HC were included in this study during the period November 2010 TO December 2011.

Serum levels of Vitamin D(25OH-D3) and calcium ,phosphate,and alkaline phosphatase were estimated.Vitamin D3 was assayed by CLIA method.

Hypocalcemia was defined as Serum calium <8 mg/dl and hypophosphatasia was defined as <4 mg/dl.Hyperphosphatasia was defined as >7 mg/dl.

Alkaline phosphatase was considered increased when level >420 IU/L.Normal level of alkaline phosphatase was taken between 150 to 420 IU/L.

Hypovitaminosis D was defined as 25(OHD)D3 <20 ng /ml.Severe vitamin D deficiency was defined as <5ng/ml,vitamin D deficiency as 5 to 15 ng/ml,vitamin D insufficiency as 15 to 20 ng/ml and vitamin D sufficiency as >20 ng/ml.

Eligible babies with defined criteria were registered for this study after obtaining the informed parental consent.

Using a standard data extraction form ,information abstracted from the charts regardind age ,sex and percentile body weight and length.

Baby data such as birth weight ,present weight ,feeding history and multivitamin supplementation were enquired and recorded.

Babies screened for underlying systemic illness.

Babies screened for any clinical evidence of rickets.

Sunshine exposure,pigmentation of the baby,residing in urban area with air pollution and living in tall buildings with sunscreen use noted.

PROFORMA FOR EVALUATION OF HYPOVITAMINOSIS D IN EXCLUSIVELY BREASTFED BABIES

Study No :

IP/OP No :

Name :

Age/sex :

Address :

Contact no :

Socio economic status :

Percapita income per month :

Birth weight :

Present weignt :

Length :

Head circumference :

Term/preterm :

Multivitamin drops : Yes/No

Artificial feeding	:	Yes / No
Underlying systemic illness	:	Yes/No
Drug intake	:	Yes/No
Skin pigmentation	:	Dark/Fair
Urban/Rural area	:	
H/O sun shine exposure	:	if yes _____mts/hrs per day
H/O covering of baby when outside	:	Yes/no
H/O living in tall buildings	:	Yes/No
H/O failure to thrive	:	
H/O protruding abdomen	:	
H/O listleness	:	
H/O seizures	:	
H/O Widening of wrist and ankle	:	
H/O stridor	:	

Physical Examination :

Lab data

a.serum vitamin D3 :

b.serum calcium :

c.serum phosphate :

d.serum alkaline phosphatise :

e.X ray wrist and ankle :

TREATMENT :

FOLLOW UP :

RESULTS

40 babies beyond the neonatal period on exclusive brastfeeding, term, appropriate for gestational age were enrolled in the study. Age and gender distribution were as follows

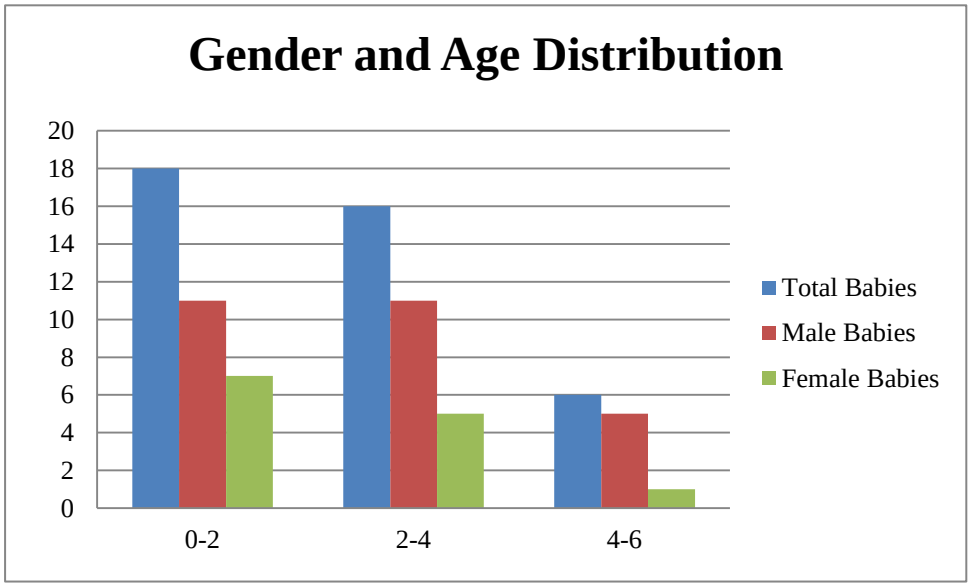


Fig 1.Gender and Age Distribution

Majority of our babies were between 1 to 2 months followed by 40% in the age group between 2 to 4 months while 15% belonged to 4 to 6 months. Out of 40 babies 28 were males and 12 were females

TABLE 1:ANTHROPOMETRIC DATA REGARDING WEIGHT FOR AGE*

AGE	NORMAL	< - 3 SD(Z-score)	-3 to -2 SD
1-2 months	18	0	0
2-4 months	16	0	0
4-6 months	6	0	0

*weight for age according to WHO growth standards

Table 1 : Describes the nutritional status of the babies enrolled in this study with regard to weight for age. All babies of < 6 months of age were of normal nutritional status.

Table 2: ANTHROPOMETRIC DATA REGARDING LENGTH FOR AGE*

AGE	NORMAL	I^0 Stunting	II^0 Stunting	III^OStunting
1-2 months	18	0	0	0
2-4 months	16	0	0	0
4-6 months	6	0	0	0

*length for age according to waterlow's classification

Table 2 depicts the nutritional status of children in this study with regard to length for age wherein all babies were well nourished.

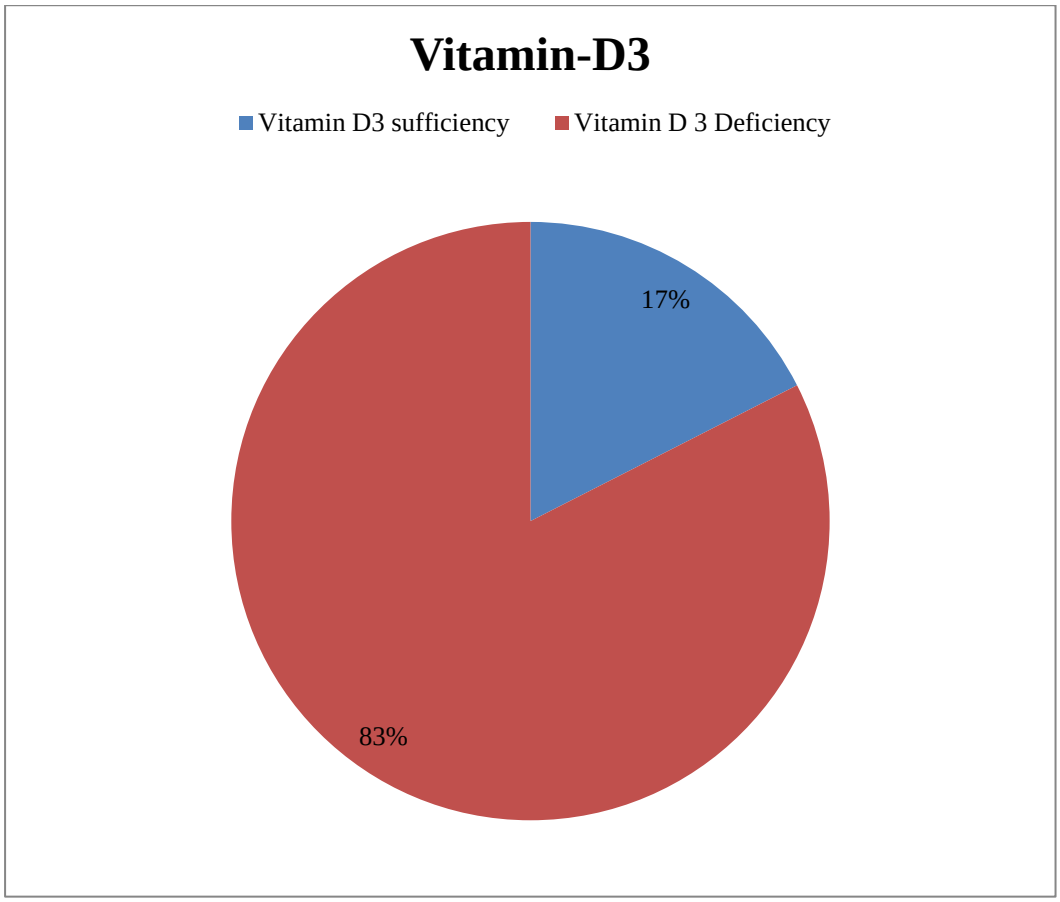
Table 3: ANTHROPOMETRIC DATA: WEIGHT FOR LENGTH*

AGE	NORMAL	MILD WASTING	MODERATE WASTING	SEVERE WASTING
1-2 months	18	0	0	0
2-4 months	16	0	0	0
4-6 months	6	0	0	0

*weight for length according to Mc Laren's classification

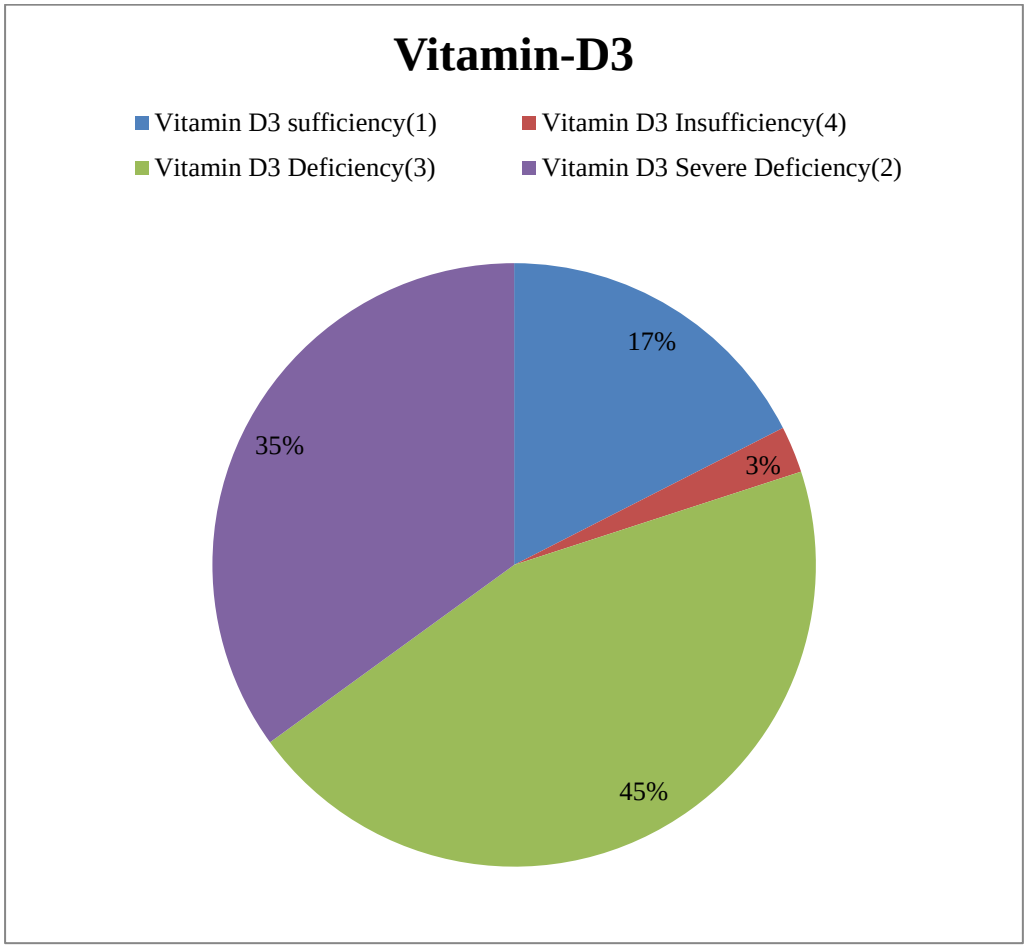
Table 3 depicts the nutritional status of the babies with regard to weight for length wherein all babies were well nourished.

Fig 2 :PREVALENCE OF VITAMIN D DEFICIENCY



In our study 33 out of 40 babies were found to be vitamin D deficient . It was around 83% of babies were vitamin D deficient and 17% were vitamin D sufficient.

Fig 3 :DEGREE OF VITAMIN D DEFICIENCY



In our study out of 33 babies with hypovitaminosis D degree of deficiency as follows

Severe deficiency – 35%

Deficiency – 45%

Insufficiency – 3%

HYPOCALCEMIA

In our study among babies with vitamin D deficiency 13 were found to be hypocalcemic. Remaining 20 babies were normo calcemic. One baby with hypocalcemia due to vitamin D deficiency presented with refractory seizures and all other babies were had only asymptomatic hypocalcemia.

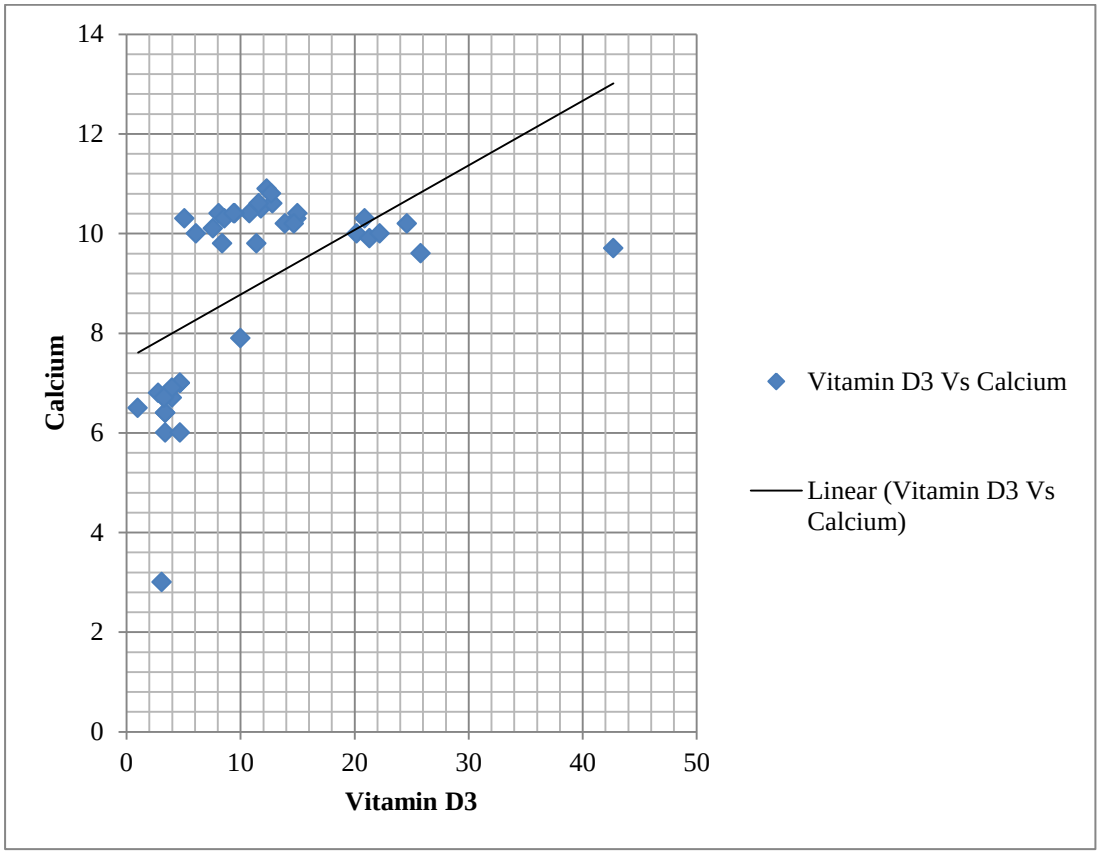


Fig 4.

Phosphate level found to be normal in all babies

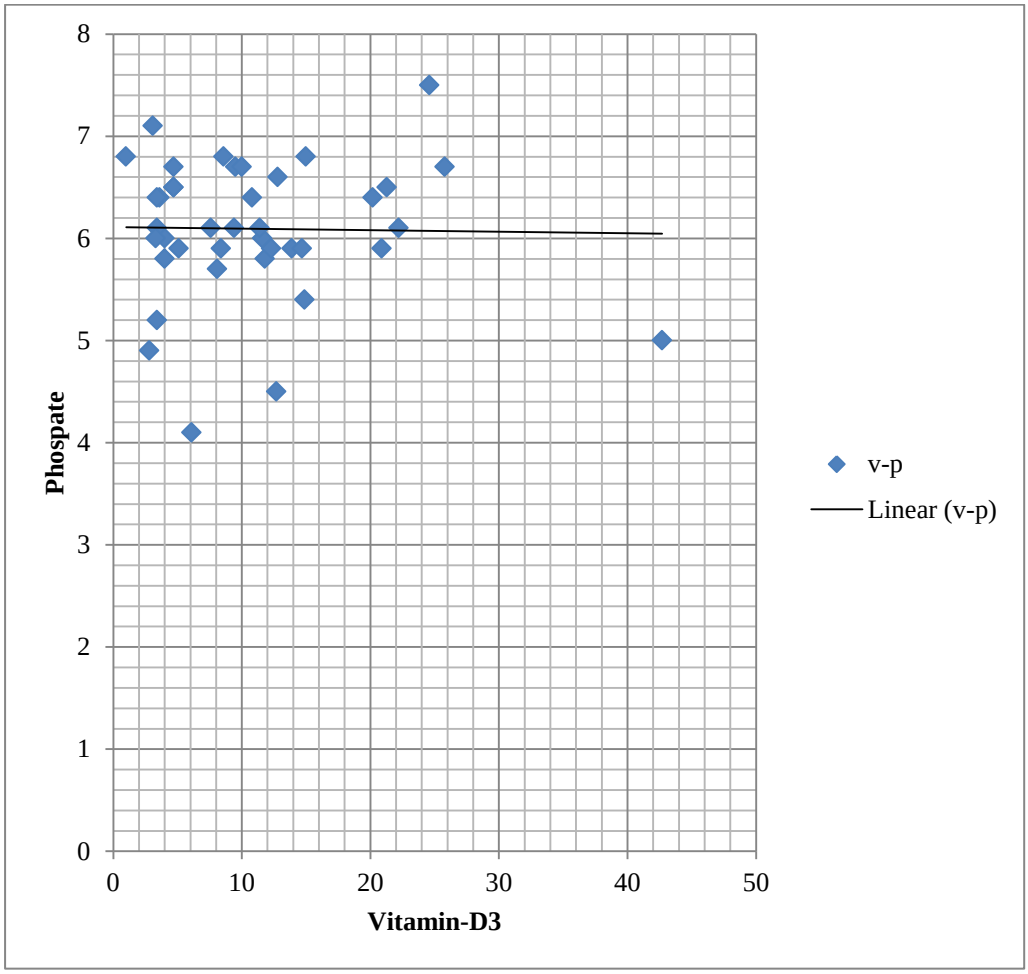


Fig 5.

Alkaline phosphatase

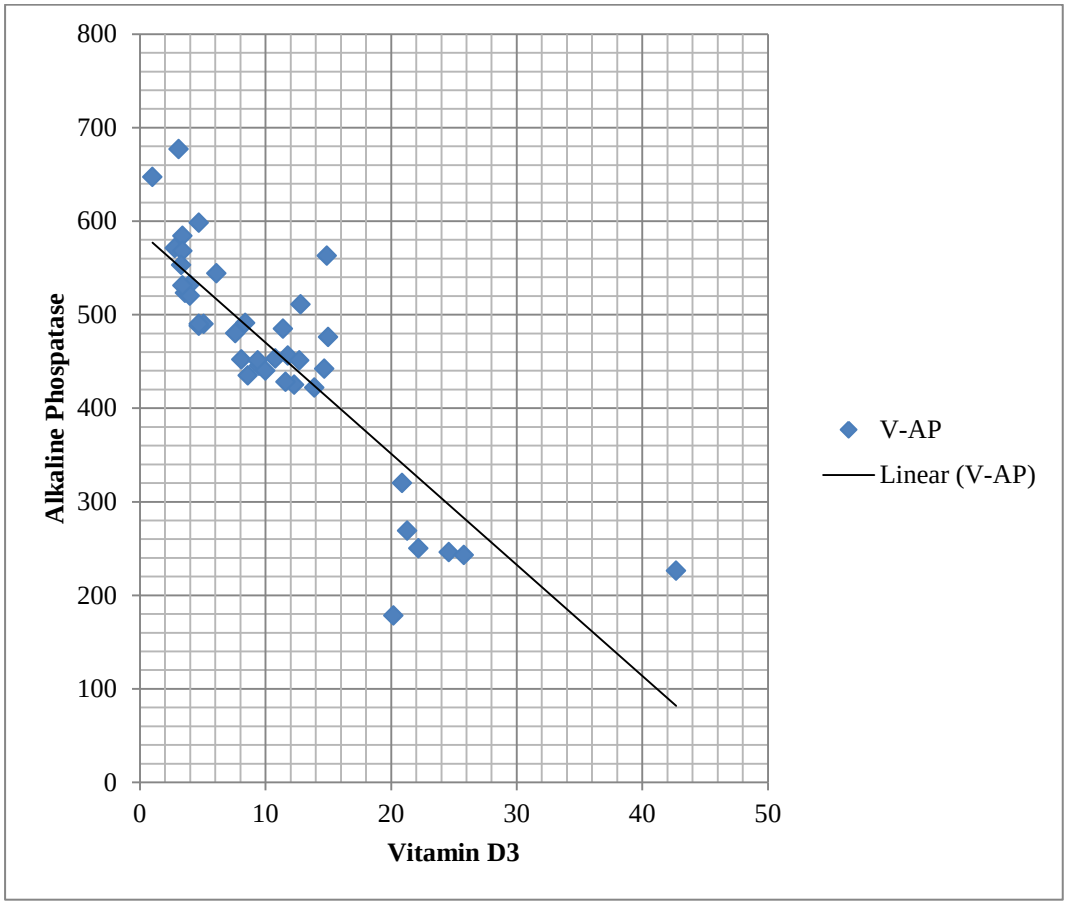


Fig 6:

In our study all babies with hypovitaminosis D had elevated alkaline phophatase level

DATA REGARDING CLINICAL PRESENTATION

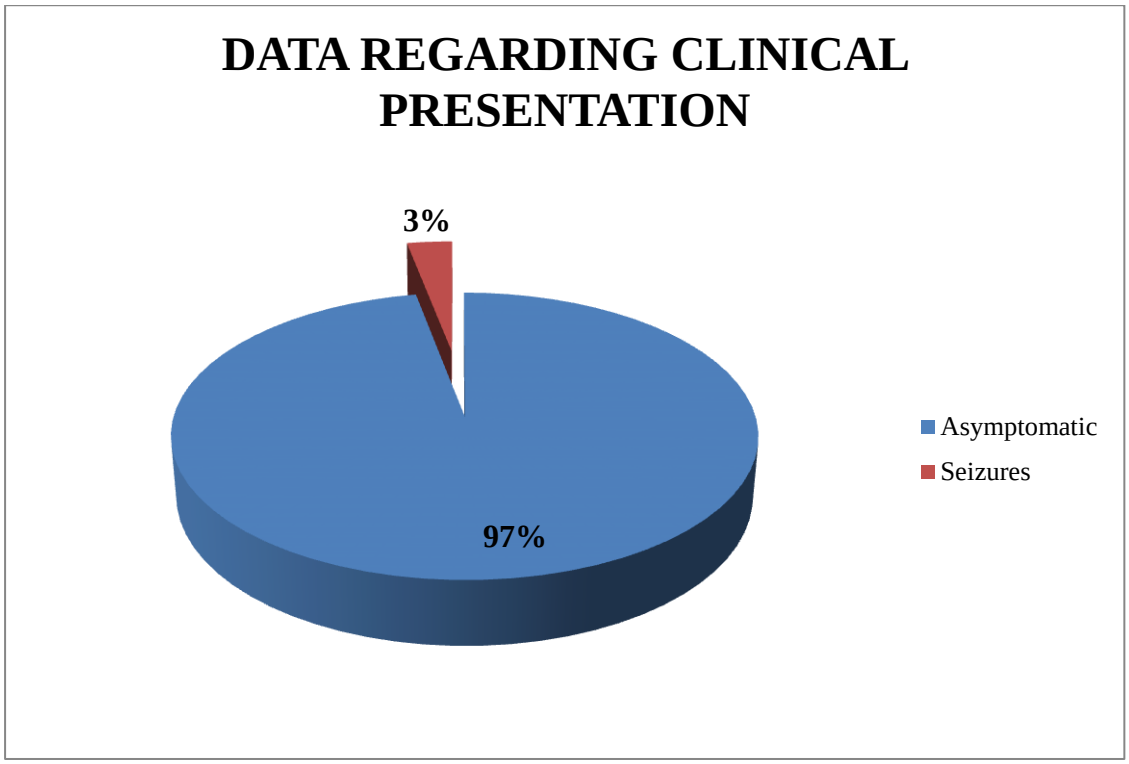


Fig 7.

1. Seizures
2. Tetany
3. Bony deformities
4. Failure to thrive
5. Asymptomatic

In our study one baby with hypocalcemia due to vitamin D deficiency presented with seizures.

All other babies with hypovitaminosis D were asymptomatic

SUNSHINE EXPOSURE

In our study all the babies had minimal sunshine exposure

Sunshine exposure score between 0 to 12

SKIN PIGMENTATION

In our study skin pigmentation follows

Table 4:

Number of fair skin baby	29
Number of dark skin baby	11

Babies with dark skin pigmentation degree of vitamin D deficiency was high.

Babies with fair skin pigmentation vitamin D deficiency relatively low.

TABLE 5: STATISTICAL DATA OF BIOCHEMICAL INVESTIGATIONS IN EXCLUSIVE BREAST FED BABIES

Parameteres	Vit D_3 25 OH ng/ml	Calcium mg/dl	Phosphorus mg/ml	ALP IU/l
Mean	10.02257143	8.81714286	6.12857143	464.97143
Range	24.8	7.9	3	434
Std.Deviation	8.346150806	1.95814538	0.68085542	116.00719

Table 5 depicts the biochemical investigations of babies who had hypovitaminosis D. Levels of alkaline phosphatise was elevated (Mean ALP 464.97 IU/L) in all of them.

Phosphate level found to be normal in all babies.

Serum 25 OH vitamin D3 levels were low (Mean 10.02 ng/ml)

Mean calcium was 8.81 mg/dl

Radiological features of rickets like fraying of ends of long bones in

X-ray of both knees and wrist were noticed in 3 babies although clinical features of rickets were absent in all babies.

TREATMENT

All these babies were treated with oral vitamin D 400 IU/day with oral calcium supplements (50mg/kg/day) were administered for 3 months in all of them. Normal levels of alkaline phosphatase for the age were achieved over a mean of 3 months.

DISCUSSION

- In our study 83% of exclusively breast fed babies are vitamin D deficient similar to report by Atiq et al. A male preponderance was noticed in our study, the reason for which is not clear.
- In our study all exclusively breast fed babies with hypocalcemia and vitamin D deficiency had inadequate exposure to sunlight as reported by Gunsel Kutluk et al[39].
- Since vitamin D is principally derived from the action of sunlight on exposed skin, inadequate exposure to sunlight, leads to hypovitaminosis D which must be made up from dietary sources. In exclusive breast fed babies the content of breast milk vitamin D also low, so vitamin D deficiency is aggravated[39]. Deficiency must be present for many months before clinical rickets becomes obvious[39]. In our study, all babies with vitamin D deficiency as evidenced by low levels of 25 OH vitamin D3 but clinical features of rickets were absent in all of them.
- Babies on exclusive breast feeding with vitamin D deficiency may have normal levels of calcium or low level of calcium

depends upon the stage of deficiency as similar to earlier report by Vanthana Jain et al[46].

- Vitamin D deficiency in exclusive breast fed babies may present with hypocalcemia seizures as similar to earlier report by SBS, et al[1].
- Nutritional rickets still remains one of the most common health problems in children in developing countries and in some regions of developed counties too, with increasing incidence in the latter[39].
- It is unfortunate that this natural source of vitamin D is not being utilized in tropical countries like India where Nature is kind enough to provide sunlight. Gessner et al recommended adequate exposure to sunlight to maintain serum 25-hydroxy Vitamin D concentration above the lower limit of the normal range[16].
- Exclusive breastfeeding is recommended upto 6 months with all its beneficial effects on child survival. However reports of biochemical evidence of vitamin D deficiency in breastfed infants both in developing and developed countries have been periodically appearing in medical literature.

- In a study conducted in the neighboring nation Pakistan[21] high prevalence of Vitamin D deficiency in breastfed infants was observed wherein out of 65 cases studied, 55% of infants were found to have low levels of Vitamin D (25 OH D3). In our study 83 % of 40 infants tested had hypovitaminosis D.
- In a study conducted in UAE by Dawodu et al 82% of 78 infants tested had hypovitaminosis D. In our study also 83% of 40 infants tested had hypovitaminosis D.
- In a study conducted in New Delhi by Bhalala et al 80% of exclusive breastfed infants were vitamin D deficient at 3 months of age. In our study 83% of exclusive breastfed babies were vitamin D deficiency.
- Greer, Marshal et al reported that vitamin D unsupplemented breastfed infants had no evidence of vitamin D deficiency during the first 6 months of life[37]. However based on observations in Alaska, Gressner BD et al concluded that exclusive breastfeeding is risk factor for vitamin D deficiency in early infancy [16].
- The American Academy of Pediatrics has recommended that 400 IU of vitamin D to be given all exclusive breastfed babies

from day one of life(36)). In the UK, the Department of Health recommends 400 units for the pregnant and lactating women, 340 units for infants under 6 months[41].

- Recommendations for Vitamin D supplementation of all exclusively breastfed infants at an early age and continuation of supplementation throughout the duration of breastfeeding have been made from observations in another study from USA[22] Our observation suggest that subclinical hypovitaminosis D widely prevalent in exclusive breastfed babies and may present with hypocalcemic seizures. Our study also emphasizes the importance of adequate exposure of sunlight to pregnant women, lactating mothers and infants and also the need for vitamin D supplementation to exclusively breast fed infants.
- To conclude there is need for global perspective for the current status of Vitamin D deficiency worldwide especially in exclusive breastfed babies and the need for policies regarding vitamin D supplementation to all exclusive breastfed babies. There are also recommendations for resetting RDA fir vitamin D intake as emphasized by Whiting et al[44].

- Reports showing increasing rates of rickets due to insufficient exposure to sunlight and inadequate Vitamin D intake are the cause for serious concern as concluded by Molgaard et al[38]. So there is an emphasis on the fact that adequate exposure to sunlight is recommended for the infants and also the pregnant and lactating mothers.
- Also we would propose to conduct nationwide study to detect subclinical hypovitaminosis D in exclusive breastfed babies and need for implementation of the national recommendations on vitamin D supplementation to exclusive breastfed babies to prevent associated co-morbidity of vitamin D deficiency.

SUMMARY AND CONCLUSIONS

1. Vitamin D deficiency in exclusive breastfed babies not only present in the developed countries, but also in developing countries like India where abundant sunshine exposure is present.
2. High prevalence of subclinical hypovitaminosis D is present among exclusive breastfed babies.
3. Vitamin D deficiency in exclusive breastfed babies can manifest as hypocalcemic seizures, if supplementation of Vitamin D is not done in them in the setting of decreased exposure to sunlight.
4. Rickets is the late manifestation of vitamin D deficiency usually after 6 months of age. It should be diagnosed in early stages before 6 months of age by assaying 25OH vitamin D3 in exclusive breastfed babies.
5. Recent studies concluded that adequate intake of vitamin D in exclusive breastfed babies cannot be met with human milk as the sole source of vitamin D. Adequate level of vitamin D in pregnancy will give adequate levels in newborn for 8 to 12 weeks only. Also vitamin D content of breast milk is low (12-60

IU/L) and RDA of vitamin D in exclusive breast babies is 200 to 400 IU/day. If exclusive breastfed babies not supplemented with vitamin D within 2 to 3 months of age, the risk of developing vitamin D deficiency rickets by 6 months of age is very high.

RECOMMENDATIONS

- Vitamin D supplementation and adequate exposure to sunlight of exclusively breastfed infants and their mothers should be practised to prevent vitamin D deficiency and its consequences in infancy.

- Urgent need to study the problem of vitamin D deficiency in exclusive breastfed babies in other parts of country and a National programme consisting of vitamin D supplementation is emphasized in light of the findings of our study.

LIMITATION

Our study analysed only prevalence of hypovitaminosis D in exclusive breastfed babies. Most important risk factor for vitamin D deficiency in exclusive breastfed babies is maternal vitamin D status which coud not be done due to logistical constraints.

AGE RELATED NORMS (Beyond the neonatal period)

Parameters	**Calcium mg/dl**	**PO_4 mg /dl**	**ALP IU/L**	**Vit D 25 OH ng/ml**
1 month to 6 months	8.8 to 10.8	3.8 to 6.5	145 to 420	20 to 30

*****Reference ranges for laboratory tests and procedures. John . F. Nicholson and Michael .A. Pesce. Nelson's text book of Paediatrics. 17^{th} edi. (2402 – 2411).

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குழந்தையின் பெற்றோர்/பாதுகாவலருக்கான ஆய்வு பற்றிய தகவல்

ஆய்வாளர்கள்

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தகவல் நாள் :-

Rickets எனப்படும் நோயானது இந்தியா போன்ற சூரிய ஒளி மிகுந்த நாடுகளில் 6 மாதத்திற்கு உட்பட்ட தாய்ப்பால் குடிக்கும் குழந்தைகளில் அபூர்வமானது என சமீபகாலம் வரை நம்பப்பட்டு வந்தது. ஆனால் சமீபத்திய ஆய்வுகள் இதற்கு நேர்மாறான முடிவுகளை தந்துள்ளன. உதாரணமாக UAE, China, Pakistan போன்ற சூரிய ஒளி மிகுந்த நாடுகளில் 6 மாதத்திற்குட்பட்ட தாய்ப்பால் மட்டுமே குடிக்கும் குழந்தைகளில் நடத்தப்பட்ட ஆய்வுகளில் Vitamin D அளவு இரத்தத்தில் 50% - 80% வரை குறைவாக உள்ளதாக கண்டறியப்பட்டுள்ளது. மேலும் தாய்ப்பாலிருந்து கிடைக்கும் Vitamin D ஆனது முதல் 2 மாதங்களுக்கு மட்டுமே போதுமானது என இந்த ஆய்வு முடிவுகள் தெரிவிக்கின்றன.

இந்த ஆய்வானது தாய்ப்பால் மட்டுமே குடிக்கும் உங்களது குழந்தையின் இரத்தத்தில் Vitamin D அளவு எவ்வளவு உள்ளது என்பதை கண்டறிய நடத்தப்படுகிறது. இந்த ஆய்வினால் எந்தவித தீங்கோ பக்க விளைவுகளோ இல்லை என உறுதி அளிக்கபடுகிறது. மேலும் இந்த ஆய்வில் உங்கள் குழந்தைக்கு இரத்தத்தில் Vitamin D அளவு குறைவாக உள்ளது என கண்டறியப்பட்டால் அதற்கான சிகிச்சைகளும் மேற்கொள்ளப்படும் என தெரிவிக்கப்படுகிறது.

நம்பகத்தன்மை (Confidentiality):-

தானாக முன்வந்து ஆய்வில் பங்குபெறும் குழந்தைகளை பற்றிய விவரங்கள் அனைத்தும் ரகசியமாக சட்டப்படி பாதுகாக்கப்படும். இந்த ஆய்வை நடத்துபவர்களும், நீதிநெறி குழு உறுப்பினர்களும் இந்த மருத்துவமனையை சேர்ந்த மருத்துவர்கள் மற்றும் ஊழியர்களும் உங்கள் குழந்தையை பற்றிய விவரங்களை தெரிந்து கொள்ள வேண்டுமானால் குழந்தையின் பதிவு செய்யப்பட்ட படிவங்கள், மாதிரிகள் பெற்று தொடர் ஆய்வின் கோப்புகள் மூலம் தெரிந்து கொள்வார்கள்.

செலவுகள் மற்றும் நஷ்டஈடு

பல பரிசோதனைகள் இலவசமாக நமது மருத்துவமனையில் செய்யப்படும் சில பரிசோதனைகள் ஆய்வாளரால் இலவசமாக செய்து கொடுக்கப்படும். உரிய வைத்தியமுறையும் பரிந்துரைக்கப்படும்.

இந்த ஆய்வு குறித்து எப்போது என்ன சந்தேகம் ஏற்பட்டாலும் உடனடியாக அது பற்றிய சரியான விளக்கம் தரப்படும்.

தவல் அளிக்கப்பட்ட ஒப்புதல் படிவம்

ஆறு மாதத்திற்குட்பட்ட தாய்ப்பால் மட்டுமே குடிக்கும் எனது குழந்தைக்கு Vitamin D அளவு இரத்தத்தில் எவ்வளவு உள்ளது என்பதற்காக இந்த ஆய்வு நடத்தப்படுகிறது என்று மருத்துவரால் தெரிவிக்கப்பட்டது. இந்த ஆய்வுபற்றி எனக்க விளக்கமாக எனது தாய்மொழியில் தெரிவிக்கப்பட்டது. இந்த ஆய்வில் பங்கெடுத்துக் கொள்வதால் எனது குழந்தைக்கு ஏற்படக்கூடிய அபாயங்கள் மற்றும் நன்மைகள் பற்றி எனக்கு விவரமாக தெரிவிக்கப்பட்டது. இந்த ஆய்வில் எனது குழந்தையை பங்கெடுத்துக் கொள்ள முழுமனதுடன் சம்மதிக்கிறேன். கேள்விகள் கேட்பதற்கு எனக்கு வாய்ப்பளிக்கப்பட்டது.

இந்த ஆய்விலிருந்து கிடைக்கும் முடிவுகளை பயன்படுத்துவதை கட்டுப்படுத்தாமலிருக்க நான் சம்மதிக்கிறேன்.

குழந்தையின் பெயர்
குழந்தையின் பெற்றோர் /
பாதுகாவலரின் பெயர்
குழந்தையின் பெற்றோர் /
பாதுகாவலரின் கையெழுத்து
தேதி
எழுதப்படிக்கத் தெரியாத பெற்றோர் /
பாதுகாவலரின் கைவிரல் ரேகை
சாட்சியின் பெயர்
சாட்சியின் கையெழுத்து
தேதி
ஆய்வாளர் / ஆய்வு மருத்துவர் பெயர்
ஆய்வாளர் / ஆய்வு மருத்துவர் கையெழுத்து தேதி

INFORMED PARENTAL CONSENT FORM

PREVALENCE OF HYPOVITAMINOSIS D IN EXCLUSIVELY BREASTFED BABIES

INVESTIGATORS NAME: Dr.V.PRABU

(To be read to care takers / parents in the presence of a witness)

Vitamin D deficiency and rickets in India has only recently received the attention as there has been a general belief that vitamin D deficiency is uncommon problem in exclusively breastfed babies due to abundant sunshine exposure. But recent studies conducted in tropical countries like china, UAE and pakistan says that 50- 82% of exclusive breastfed babies are vitamin D deficient. Also these studies proved that breast milk vitamin D alone is not sufficient for exclusively breastfed babies.

Vitamin D deficiency without any clinical manifestations is the earliest stage of rickets. So by doing vitamin D assay in exclusively breastfed babies(less than 6 months) we can diagnose rickets in early stages even before 6 months of age.

How is the study being done?

The study will be conducted in babies upto 6 months of age whom are exclusively breastfed. The doctor will ask questions and examine the child to make sure that it is safe for him/her to enter the trial. All babies subjected to blood investigations according to manoeuvre protocol which will be done under strict aseptic precautions only.

Can I refuse to join the study?

You may refuse to participate or withdraw from this study anytime.

Is there any benefit or harm?

Appropriate therapy can be advised based on this study details. The disease can be diagnosed earlier. It may be possible to reduce the severity of the

disease by appropriate therapy. No additional harm will be caused by this study.

Confidentiality:

The data collected from the study will be used for the study purpose only. The results of the study are to be published in medical journals. Personal of the children participating in the study will be kept confidential. There will not be any disclosure about your child's information without your attention.

Cost & compensation:

The investigations warranted will be done at free of cost by the investigator. Any questions that may arise at any point of time will be willingly clarified.

1) I have read information sheet regarding study to be conducted,& have fully understood the information given herein. Further questions have been answered & doubts fully clarified.
2) In fully understanding & with a free will, I hereby consent to include my child in the study. I also understood that I may withdraw my child from the study at anytime without stating any reason.
3) I understood that the investigators and researchers involved in the study, members of ethical committee, the doctors & employee of this hospital will be granted access to the data collected without my consent.
4) I agree that I will not prohibit the use of collected data & measured outcomes of this study.
5) I wholeheartedly consent for my child to be included in this study.

Sign of investigator & date

Sign of the parent/ Guardian& date

Name:

Sign of witness & date

Relation to the child: