AIN SHAMS UNIVERSITY
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MEDICAL AND PSYCHOMEDICAL DEPARTMENT

STUDIES ON RICKETS AND ITS EFFECTS ON HEALTH AND GROWTH OF PRE-SCHOOL CHILDREN

THESIS

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STUDIES

BY

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THANKS

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INTRODUCTION

AND

AIM OF THE WORK

INTRODUCTION

Rickets is a systemic metabolic disease resulting from some sort of disturbance in calcium-phosphorus metabolism, characterized pathologically by defective mineralisation of bone and clinically by various skeletal , muscular, and neurological manifestation (GREENFIELD, 1975). According to this definition rickets is considered as a nutritional problem related to many other aetiological and socio-economic factors causing undernutrition in Egypt . (KHALIL , 1970).

Generally, it occurs during the first two years of life when the growth is rapid . The cause of rickets is a deficiency in vitamin "D", brought about by the failure to expose the skin to the ultra-violet rays of the sun . Rickets is responsible for bowed legs, knock knee , flat foot , and is associated with rachitic rosary and pigeon chest . Deformity of pelvic bones produced by rickets during childhood , is one of the causes of difficult labour and may result in fetal or maternal death .

Rickets is most common in cities where children's exposure to sunlight is limited . In high latitude, rickets is most common in winter when the hours of daylight are the shortest . Rickets does not appear in the mortality tables because it is seldom a direct cause of death .

The full-term infants should receive 400 IU daily of vitamin "D", and this should be continued throughout child-hood ,while pre-mature infants require larger doses .

Vitamin "D", fortified milk , (which is now available in most cities and contains 400IU of vitamin "D", per quart) , is one of the most useful forms of mass-medication in preventive medicine .

Nutritional osteomalacia in the adult is the counterpart of infantile rickets .(MAXCY , 1980) .

AIM OF THE WORK

The study briefly describes the effect of rickets on health and growth of pre-school children , also, studies the relationship between rickets and the type of infant's feeding (breast or artificial feed).

On the other hand , this study aims at describing the relationship between rickets and other nutritional and infectious diseases affecting children .

This study will also, put forward into light the various factors that play a role in causing rickets .

REVIEW OF LITERATURE

HISTORY

HISTORY

The first full description of rickets was published in a treatise by Francis Glisson , in 1650, an English professor at Cambrige . The idea that rickets is connected with nutrition began to develop in the last quarter of the 19 th Century .

In about 1890 , Palm , an English medical man , first pointed out that rickets is prevalent where there is little sunlight and quite rare wherever sun-shine is abundant . He recommended the use of sunlight in treatment of rickets .

In 1919 -1920 , Mallenby ,& Huldschinsky , reported that ultraviolet light cured rickets in children.

In 1848 , cod liver oil was recommended for the treatment of adult rickets and later for rickets in children . (WILSON , 1971).

In 1924 , STEENBOCH & HISS , were able to show that many plants acquire vitamin "D" activity after exposure to ultraviolet irradiation .(MAZURE , 1971).

In Egypt , RUFER , 1921 , reported in his studies that , statuettes and paintings found in tombs of ancient Egypians showed that , bow legs typical of rickets were observed repeatedly since it has been existed for a period of at least 5000 years .

Evidence of rickets was also found in the skeleton of the stone age by KJERULF , 1922 .

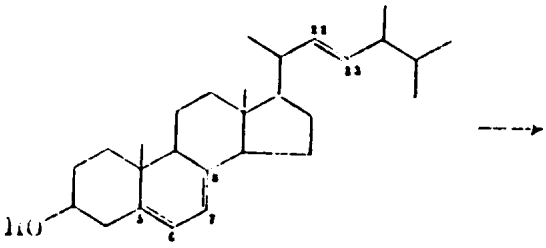
VITAMIN "D" :

- CHEMISTRY :

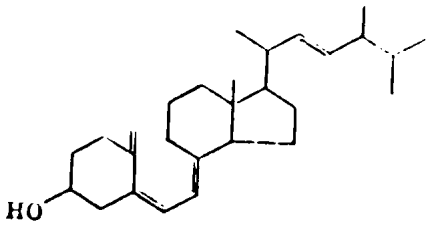
Vitamin "D" , is a group of compounds , all of them are steroid in nature (sterol) , and found mainly in animals . Vitamin "D" is derived from a certain precursors , only two of them occur in nature :

1- Ergosterol : producing vitamin "D2" . (calciferol).

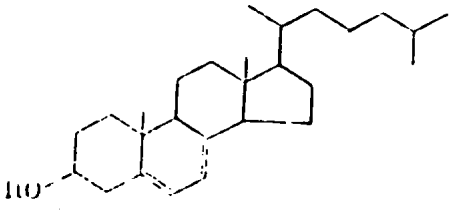
2- 7-Dehydrocholesterol : producing vitamin "D3".(cholecalciferol)
(MAZUR , 1971).



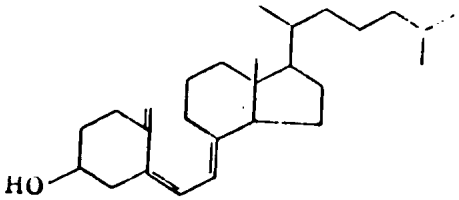
Ergosterol



Ergocalciferol (vitamin D_2)



7-Dehydrocholesterol



Cholecalciferol (vitamin D_3)

The natural source of vitamin "D" , for the humanbeing is the chole-calciferol (vitamin D 3) , which is produced in the skin by exposure to ultra-violet rays of sunshine which activate the skin 7 - Dehydrocholesterol , (BARNETT, 1968) . Since this vitamin is formed in the skin and acts on distant organs (gut & bones) , LOOMS ,1967 suggested that it could be classified as a hormone rather than a vitamin . The rate of synthesis in the skin is determined by the degree of exposure to ultra-violet rays and the degree of pigmentation , as a heavily pigmented child is more prone to have rickets than a fair child .

Neither cow's milk (0.3 to 4 U / 100 ml) , nor human milk (0 to 10 U / 100 ml) , ordinarily contains appreciable quantities of vitamin " D" . (BARNETT, 1968) .

PROPERITIES :

1- Vitamin "D" is soluble in fat solvents .
2- It is resistant to oxidation and to heat .
3- It can form esters with fatty acids since it is alcohol , (OH group at carbon 3) .

DAILY REQUIREMENTS :

- 400 - 800 IU / day for infants .

- 400 IU / day for adults .
- 800 IU / day for pregnant and lactating mothers . (CODEX, 1979 .

$ N.B The International Unit (IU), of vitamin D is the activity of 0.025 Ug of crystalline vitamin D2 (calceferol) .

SOURCES OF VITAMIN D :

Vitamin "D" , itself is widely distributed in animals e.g fish , mainly in their liver (cod liver oil) ,(halibut liver oil) , egg yolk is very rich in vitamin D3 . Milk is a poor source of vitamin D3 . Human milk contains negligible amounts , about (0 - 10 U / 100 ml) . BARNETT, 1968 . Vegetables & fruits are poor sources of vitamin D; (KHALIL, 1970) .

Ultraviolet irradiation is essential for activation of skin 7 Dehydrocholesterol to vitamin D3 . (GOODMAN 1970) .

ABSORPTION OF THE VITAMIN :

The ingestion of vitamin "D" requires the presence of the bile for its absorption . (GREAVES & SCOMIDET ,1933.) Vitamin "D" absorption is decreased in chronic diarrhea, steateorrhea & biliary obstruction or insufficiency .

After absorption from gut , most of the vitamin appears in the lymph as lipoprotein complex (SCOCHTER , et'al, 1964) . Then both vitamin D2 & D3 are hydroxylated in the liver to 25, hydroxycholesterol (25 , H.C.C) ,this is the form in which they circulate in the blood and is further hydroxylated to 1,25 , dihydroxycholesterol in the kidney and this is the active metabolite . (EDEIKEN , 1973.

STORAGE :

- The liver is the main site of storage . (MASEK , 1969).
- Vitamin "D" ,is found in bones , intestine , kidney , blood , muscles and skin .

EXCRETION :

- By bile to be reabsorbed by small intestine .
- By milk .
- No unchanged calciferol is excreted in the urine .(CODEX, 1979) .

PHYSIOLOGY: OF VITAMIN "D" :

Vitamin "D", the antirachitic vitamin , is essential for utilization of calcium and phosphorus .

Vitamin "D" is necessary for :

1- Absorption of diatery calcium and phosphorus from the intestinal tract .

2- Reabsorption of phosphate by renal tubular cells (an- interaction with parathyroid hormone) .

3- Maintenance of serum - calcium level by mobilizing calcium from bone when the oral intake is inadequate (an interaction with parathyroid hormone , (SLOBODY, 1968) .

Normal calcium and phosphorus metabolism is dependent on proper levels of vitamin "D" . Blood levels of these ions are influenced by gastrointestinal absorption , skeletal metabolism , and renal excretion and are predominantly under the control of vitamin "D" , parathyroid hormone and thyrocalcitonin .

The concentration of ionized calcium is also, dependent upon blood PH , concentration of plasma protein .(HARRISON, et'al,1962) .

VITAMIN "D" DEFICIENCY :

Two aetiologic factors are responsible for vitamin "D", deficiency rickets . These are dietetic factor and inadequate exposure to sunlight . Infants receiving breast milk alone may have low circulating levels of vitamin "D", and a much increased incidence of rickets , when not exposed to sun . (LACK , AWALA , 1977) ; .

Undernourished children are less active and would not creep outdoors to be exposed to ultraviolet rays . These rays are considered the 2nd factor responsible for the activation of vitamin "D" and its deficiency leads to rickets . Deficiency of vitamin "D" results in an inadequate absorption of calcium and phosphorus . The failure to maintain calcium balance with the resulting mobilization of calcium from skeletal stores , represents a much more serious disorder during the period of skeletal growth .(JONIXIS & HUISMAN, 1953) .

Consequently , vitamin "D" deficiency in infants and children rapidly leads to serious metabolic bone disease known as rickets . In adults , only small amounts of calcium are necessary to maintain balance . If calcium absorption is

seriously impaired or if its requirement are increased , as in pregnancy or lactation , lack of vitamin "D" can lead to osteomalacia (adult rickets) . (JONIXIS& HUISMAN, 1953) .

The net result of lack of vitamin "D" , is a decrease in the concentration of both calcium and phosphorus ions in extracellular fluid , so that , the ionic concentration is inadequate for mineralization of bone matrix . The deficiency of phosphorus ion is more critical , and if the hypophosphataemia is marked , the serum calcium ion concentration may remain in the normal range , because of markedly decreased uptake of calcium phosphate by bone . A secondary effect of calcium and phosphate deficiency is an over proliferation of osteoblasts , which leads to the formation of weak bone .

The elevated alkaline phosphatase activity in serum in vitamin "D" deficiency ,represents release of this enzyme from osteoblasts. Hypocalcaemia of vitamin "D" deficiency is not only due to poor absorption of calcium from intestine, but also due to a failure of response of the bone cells to parathyroid hormone . An additional manifestation of vitamin "D" deficiency is generalized amino aciduria due to abnormality of renal tubular reabsorption of amino acids(BAKNETT,1968).

RICKETS :

Rickets is a term applied to an abnormality of growing bones related to lack of vitamin "D", lack of alkaline-phosphatase and imbalance of plasma calcium & phosphorus ratio .

There is a range of causes for such abnormality which are:

1- NUTRITIONAL VITAMIN D , DEFICIENCY :

   a) Congenital .
   b) Prematurity .
   c) Infantile .
   d) Toddler .
   e) Adolescent .

2- MALABSORPTIVE STATE .:

   a) Celiac disease .
   b) Obstructive jundice .

3- HYPOPHOSPHATASIA .

4- HYPERPHOSPHATURIC & HYPOPHOSPHATAEMIA .

5- RENAL RICKETS .

6- DRUG INDUCED .

   e.g Phenobarbitone , phenytoin .

7- PSEUDO-VITAMIN D DEFICIENCY RICKETS .

1- NUTRITIONAL VITAMIN "D" DEFICIENCY :

a) CONGENITAL RICKETS :

Mainly occur in the neonatal period and caused by maternal osteomalacia due to high extract flour in diet, less sunshine and inadequate vitamin "D" supplements . This type of rickets is often complicated with neonatal tetany. (FORFAR, 1973) .

b) PREMATURE RICKETS :

It occurs in the period between one month and 6 months of age . Presents by craniotabes away from suture and caused by preterm delivery with failure to give adequate vitamin "D" after birth , (800 IU) daily. (FORFAR, 1974) .

c) INFANTILE RICKETS :

It occurs between 6 - 12 months of age when the child is not yet able to walk . The most present features are generalized convulsion , costochonderal beading , bow legs , kyphosis and slow fontanelle closure . (FORFAR, 1978) .

d) TODDLER RICKETS :

This is a nutritional rickets occuring in the period

between one year to four years of the child ' s age , the disease presents by marked bow legs and coxa vara . (ARNEIL ,1963) also, delayed closure of fontanelle and costochondral beading . (GAVIN. , 1973).

e) ADOLESCENT RICKETS:

This nutritional rickets occurs in the period of age of the child between 8 years to 16 years and the more common signs are :

- Coxa vara , genu valgum .
- Waddling gait .

This type is caused by poor intake of vitamin "D" diet . so a daily dose of 300 IU calciferol would seem a good propnylactic measure . (GAVIN , 1973) .

2- MALABSORPTIVE DISEASE :

In celiac syndrome , cystic fibrosis of pancreas, and atresia of the bile ducts , there are poor absorption of vitamin "D" and loss of calcium and phosphorus in faeces . The amount of blood calcium may be diminished resulting in a tendency toward tetany . The treatment is ultraviolet irradiation , and large doses of vitamin "D" , .(SLOBODY, 1968) .

3- HYPOPHOSPHATASIA :

A familial disease inherited as an autosomal recessive trait and characterized by failure of calcification of bone matrix . There are premature loss of teeth and decrease or absence of alkaline phosphatase enzyme .(SLOBODY,1968). Secondarily to this,hypercalcaemia develops presumbly leading to vomiting , hypotonia and constipation . (FORFAR , 1978) .

4- HYPERPHOSPHATUREA :

It is inherited as a ressive trait, it usually occurs at 4 to 8 months of the child's age . There is delayed growth , frequent vomiting , fever , polyuria , dehydration and rickets .

- HYPOPHOSATAEMIA & VITAMIN D RESISTANT RICKETS :

It is an enzymatic defective inherited disease as a sex-linked dominant trait . When present , the rickets is due to an associated faulty absorption of vitamin"D" from gastrointestinal tract . Serum phosphate level is low , calcium level is normal . Alkaline phosphatase level is normal or slightly increased . Stool calcium is deminished. The condition usually becomes clinically apparent at 12 to 24 months of age .

5- RENAL RICKRTS :

It is due to renal insufficiency, secondry to chronic glomerulonephritis , congenital anomalies of the urinary tract as polycystic kidney . There are azotaemia , hyperphosphataemia hypocalcaemia , and dwarfism . Parathyroid activity is stimulated by hyperphosphataemia and hypocalcaemia . (SLOBODY , 1968).

6- DRUG INDUCED :

e.g phenobarbitone and phenytion .(CAMPION , PELC and ATKINS ,1976) .

7- PSEUDO- VITAMIN "D3", DEFICIENCY RICKETS :

It is inherited as an autosomal recessive condition occuring in boys and girls . Clinically it presents as nutitional rickets , but very large doses of vitamin "D" , usually between 250 Ug and 100 Ug (10,000 - 40,000 IU) per day are required for treatment . Such dose level must be maintained through life The disease usually presents in the first year of life with clinical feature and serum biochemical pattern similar to rickets . The serum calcium and phosphate levels are low or normal and there is increased generalized aminoaciduria . (FANCONI& PARDER , 1969) .

PATHOLOGY OF RICKETS :

New bone formation is intiated by the osteoblasts, which are responsible for matrix deposition and its subsequent mineralization . Osteoblasts secrete collagen and changes in polysaccharides, phospholipids , and alkaline phosphatase , follow until mineralization occurs in the presence of adequate calcium and phosphorus .

Defective growth of bone in rickets results from retardation or suppression of normal growth of epiphyseal cartilage and of normal calcification . Cartilage cells fail to complete their normal cycle of proliferation , degeneration and subsequent failure of capillary penetration . This occurs in a patchy manner . The result is a frayed & irregular epiphyseal line at the end of the shaft . There is also, failure of mineralization of osseous and cartilaginous matrix. As a result a wide irregular , frayed zone of non rigid tissue (the rachitic metaphysis) is produced . This zone is responsible for many of the skeletal deformities . It becomes compressed and bulge laterally , producing flaring of the ends of the bones and the rachitic rosary .

On the other hand the cortical bone is replaced by osteoid

tissue which fails to mineralize . If this process continues the shaft loses its regidity , and the resulting softened and rarefied cortical bone is readily distorted by stress , and deformities and fracture results . (NELSON,1975).

HEALING RICKETS :

With healing , degeneration of cartilage cells occurs along the diaphyseal border , capillary penetration is resumed and calcification takes place in the zone of preparatory calcification . This calcification produces a line clearly demonstrable in roentgenogram. As healing progresses the osteoid tissues between this line of preparatory calcification and diaphysis also, becomes mineralized. Also the osteoid tissue in the cortex and trabeculae of the shaft rapidly become mineralized . Months or years may be required to repair the deformities . (NELSON , 1975) .

CHEMICAL PATHOLOGY :

In healthy infants the inorganic serum phosphorus concentration is (4.5 to 6.5 mg / dl) whereas in rachitic infants it is usually reduced to (1.5 to 3.5 mg / dl) .

The serum calcium level is usually normal ,(9 to 11 mg/ dl), but under certain conditions it too is reduced to (5 to 6 mg/dl) and tetny may develop .

Calcium is necessary for normal function of nerve, muscle,and endocrine glands , therefore when its serum level is decreased as a result of vitamin "D" deficiency , the parathormone is secreted , this leads to mobilization of calcium and phosphorus from the bone . The serum calcium is thus maintained , but secondary effects occur, which include change in bone , low serum phosphorus level and elevation of serum phosphatase enzyme . (NELSON , 1975) . The serum alkaline phosphatase which in normal children ranges between (5 to 25 King-armstrong units/ dl) is elevated in rickets to 75 king-armstrong units/dl. (FORFAR , 1978) .

As rickets heals , the phosphtase level returns slowly to normal . When there is a low serum calcium , owing to deficient absorption of vitamin "D" , the parathormone secretion is increased . This parathormone lowers tubular reabsorption of phosphorus , resulting in hypophosphataemia . In addition parathormone mobilize calcium from the bone through an effect on osteocyts , leading to calcium release from mature bone . It also, acts on the kidney to reduce calcium clearance and on the intestine to increase calcium absorption .

Vitamin"D" produces its action in regulation of calcium and

phosphorus metabolism , increases intestinal absorption of calcium and phosphorus , increases renal tubular reabsorption of phosphate and it has a direct effect on deposition of calcium & phosphorus in bone . (NELSONE, 1975) .

CLINICAL MANIFESTATION OF VITAMIN D DEFICIENCY RICKETS :

After several months of vitamin "D" deficiency osseous changes of rickets can be recognized . Breast fed infants whose mothers have osteomalacia may have rickets within 2 months .

The earlist manifestations of rickets are expressed in the form of constitutional symptoms such as anoerexia, constipation , irritability , jerky movement of head , poor sleep and hypotonia . The disease is characterized by delayed movement , the young infant fails to raise his head or sit up at the normal type , and that an older infant fails to crawl or walk , and dentition is often delayed. The infant may show nutritional anaemia . Those with severe marsmus,with entire cessation of growth are unlikely to show active rickets . These cases are characterized by generalized hypotonicity of limbs and abdominal muscles, which lead to abdominal distention . Also, gastrointestinal

symptoms are common . (ELLIES , 1968) .
One of the early signs of rickets is craniotabes . Craniotabes is due to thinning of the inner table of the skull and is detected by pressing firmly over the occiput or posterior parital bones . A ping pong ball sensation will be felt .
Palpable enlargement of the costochondral junctions (the rachiti rosary) , and thikening of wrist and ankles are other early evidence of osseous changes .(BARNETT, 1968) .

In advanced rickets , signs are easily recognized and radiological signs or evidence have a certain characteristic according to the part affected as in the following :

HEAD :

Craniotabes may disappear before the end of the first year, though the rachitic process continues . The softness of the skull may result in flattening of the head . Failure of closure of the anterior fontanelle is most likely to be observed when rickets develops during the first fifteen months . The central part of the parital and frontal bones are often thickened , forming prominences or bosses which give the head a box like appearance . The anterior fontanelle is larger than normal , its closure may be delayed until

after the second year of life . (SLOBODY , 1968) . Eruption of the temporary teeth is sometimes delayed and out of the normal order . There may be defect of the enamel and extensive caries . The permanent teeth which are calcifying may be affected , usually the permanent incisors , canines and first molars, they show defect of the enamel .

THORAX :

In advanced rickets ,the most common deformities of thorax consist of :

1- Harrison's sulcus : A horizontal groove laying just above the attachment of the diaphragm , developing as a result of respiratory embarassment . (although this deformity is particularly liable to occur in the softened thorax in rickets it is not diagnostic, and may be seen in non rachitic children if severe respiratory embarassment has occured early .

2- Beading of the ribs (rachitic rosary) , due to enlargment of the costochondral junction .

3- Deformities of the sternum , which are often irregular and asymetrical . The sternum may be depressed or prominent (pigeon chest) , with lateral depression of the ribs .

4 - Flaring or upward deflection of the lower ribs .(NELSON, 1975).

PELVIS :

Deformities of the pelvis , of which reduction of the inlet is clinically most important , it may complicate delivary during the child bearing age of rachitic females. (ELLIES , 1968) .

SPINAL COLUMN :

Slight to moderate degree of lateral curvature (scoliosis) are common , and a kyphosis may appear in the dorsolumbar region in rachitic children who sit up . Lordosis of the lumbar region may be seen in erect position .

EXTREMITIES :

As the rachitic process contiues , the epiphyseal enlargment at the wrist and ankles become more noticeable. Beading of the softened shaft of the femur , tibia and fibula results in bow legs or knock knee , the femur and tibia may show an anterior convexity . Coxa vara is sometimes the result of rickets . Greenstick fractures occur in the long bones, but seldom cause clinical symptoms .
Deformities of the spine , pelvis , and legs result in reduction in height of body , (rachitic dwarfism) .

frayed and widened with appearance of haziness in the zone of provisional calcification followed by disappearance of this zone . This reflects the failure of deposition of mineral salts within the organic matrix of the degenerated cartilage cells . (AEGERTER ; 1975) . This is in contrast to the normally sharply demarcated and slightly convex ends . The distance between the distal ends of the ulna and the radius and the metacarpal bones is increased , since the large metaphysis, which is not calcified , does not appear on the roentogram . The denisty of the shaft is decreased , but the trabeculae are usually prominent . The outer cotour of radius appear double due to the formation of osteoid tissue layer .

HEALING RICKETS :

Beginning of healing is indicated by the appearance of the line of preparatory calcification. This line is separated from the distal end of the shaft by a zone of the osteoid tissue. As healing progresses and the osteoid tissue becomes calcified, the shaft grows towards the line of preparatory calcification until it becomes united with it . (MACKAY, 1975) .

DIFFERANTIAL DIAGNOSIS :

Non rachitic craniotabes is sometimes present in

the immediate post-natal period , but it tends to disappear before rachitic softening of the skull would become manifest (Second to fourth months of life) . Craniotabes also, occurs in hydrocephalus and osteogenesis imperfecta , but it is not difficult to differentiate these conditions from rickets .

- Enlargement of the costochonderal junctions in rickets ,scurvy and chondrodystrophy . The enlargements in rickets are rounded knobs , whereas in scurvy there is a ledgelike depression while in chondrodystrophy there may be irregular concave outline of the distal ends of the bones , but there is no roentographic evidence of fraying .

- Bow legs can be the result of rickets , but may be familial characteristic ;. (NELSON , 1975) .

COMPLICATION :

Respiratory infections such as bronchitis and broncho-pneumonia are common in rachitic infants . Pulmonary atelectasi is frequently associated with severe deformity of the chest . Chronic gastroenteric disturbances are common , there may be diarrhea , or constipation , or alternation of both . Anaemia due to iron deficiency or accompanying infection often develops in severe rickets . Tetany may occur as a result of hypocalcaemia . (ELLIES , 1968).

PROGNOSIS :

Though spontaneous healing of mild rickets often occurs from exposure to sunlight , severe cases require more energetic treatment . If sufficient amounts of vitamin "D" are administered healing begins within a few days and progresses slowly until the normal bone structure is restorted . In many instances the enlargement of the epiphysis, the rosary and the deformity of the skull disappear only after months or years of treatment .

In advanced cases , there may be permanent osseous alternation in the form of bow legs , knock knees , curvature of the upper arms , deformities of the chest and spine , , rachitic pelvis , rachitic coxa vara and dwarfism .

Rickets in itself is not a fatal disease , but complications as tetany , and infections as intercurrent pneumonia , enteritis and tuberculosis are more likely to cause death in rachitic than in normal children . (NELSON,1975) .

PREVENTION OF RICKETS :

Rickets can be prevented by adequate exposure to ultraviolet light and proper administration of vitamin "D". (DAVIDSON , 1969) .

Breast fed infants should receive a daily supplement of 10 microgram (400 IU) , of vitamin "D" . Supplements should be continued until at least the first birth day and then reduced or stopped if the diet is satisfactory .

- Preterm infants should receive double dose of vitamin "D", 20 microgram (800 IU) , daily for the first three months and 10 Ug (400 IU) daily thereafter . (FORFAR , 1978).

Milk for infants should be fortified with vitamin "D" . Health education to mothers to expose their children to sunlight, and give them the adequate daily requirements of vitamin "D" and also they should administer this vitamin during their pregnancy and lactation . (NELSON, 1975 ,). An epileptic child given anticonvulsant drugs should receive vitamin "D" , as a prophylactic measure (CODEX, 1979) .

TREATMENT OF RICKETS :

Natural and artificial light are effective therapeutically, but oral administration of vitamin "D" is referred . The daily administration of 1500 to 5000 IU (6 to 20 drops of a preparation containing 10,000 units per gm) will produce healing within 2 to 4 weeks except in the unusual cases of vitamin "D"-refractory rickets .

The feeding of 600,000 units of vitamin D in a single dose , and no further vitamin for several months , may be advantageous . This is followed by more rapid healing . If no healing occurs within two weeks , the dose may be repeated once . If still no healing occurs , the rickets is resistant to vitamin D .After healing is complete the dose of vitamin D should be lowered to 400 IU daily . (NELSON , 1975) .

HYPERVITAMINOSIS D :

Ingestion of excessive amounts of vitamin "D" , results in signs and symptoms similar to those of idiopathic hypercalcaemia, which may be due to hypersenstivity to vitamin "D" , symptoms develop after one to three months of large intake of vitamin D, they include hypotonia , anorexia , irritability , constipation polyuria & pallor . Hypercalcemia and hypercalcuria are notable .

Evidence of dehydration is usually present ,aortic valvular stenosis, hypertension , retinopathy and clouding of cornea may occur . The urine may contain albumin , renal damage and metastatic calcification occur . Reontogenogram of the long bones reveal metastatic calcification and generalised osteoporosis . (MACKY, 1975) .

PREVENTION OF HYPERVITAMINOSIS D :

By careful evaluation of vitamin "D" dosage .

TREATMENT :

This includes discontiuation of vitamin D intake and a decrease in intake of calcium .

For severly involved infants , aluminum hydroxide by mouth and cortisone , may be used . (FORBES , 1968) .

TETANY OF VITAMIN D DEFICIENCY (INFANTILE TETANY):

This may be regarded as a complication of rickets . Tetany of vitamin D deficiency occurs most frequently between the ages of 4 months and 3 years . The condition is essentially one of the neuromuscular hyperexcitability due to reduction of ionized calcium . When the serum calcium level falls below 7 to 7.5 mg/dl there is muscular irritability , apparently due to the loss of the inhibitory control that the ionized calcium of the serum exerts upon the neuromuscular junction . (MACKAY ,1975) .

The disease may remain latent or the onset may be fulminating . In the latter cases a generalized convulsion will be the first symptom . Spasm of the larynx will cause a high pitched sound with respiratory embarrassment. The infant becomes cyanotic during such an attack and appears about to suffocate, though death in an attack is fortunately rare .
The administration of calcium gluconate (10 to 20 ml of 10 % solution IM or IV) , and vitamin "D" is curative .
Therefore , the prognosis of this case is recognized and treated promptly . (ELLIES , 1968) .

EPIDEMIOLOGY OF RICKETS

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Rickets is declining all over the world , particularly Europe , America , yet it is still a health problem in developing countries,tropical & sub-tropical countries . (BARNETT, 1968). In Egypt , our national nutrition survey demonstrated that our children are retarted in growth , particularly liner growth . Galal, et'al , 1970 ; reported that the incidence of rickets during the first two years of life in children studied , was 11 - 12 % .

In 1975 , the preschool children from two villages have been examined for clinical signs of active & healed rickets by Kamel, et'al, 1975 , they reported a prevalence of active rickets in the first three years of life ranging between 22.0 % and 28.1% and various works showed that ricket is presented in a marasmic children .

The observation , high prevalence of rickets could be explained by quantitative deficiency of vitamin "D " in diet of infants and their keeping idoors without exposure to sunlight .

On the other hand, there is a great association of rickets and diarrhea , protein caloric malnutrition (P.C.M) , type of feeding and serial order of the child . (SHUKRY ,1973)

Mothers play a role in the development of (P.C.M), by

their ignorance and reluctance in seeking easily medical advice, be responsible for development of rickets.(BARNETT, 1968). Generally , some contributary factors were noted which determine the degree of rickets , these are :

- AGE INCIDENCE :

It is more frequent in the second half of the first year and the first half of the second year of life , where the disease manifest itself when the growth is rapid . Premature infants are more exposed to the disease and this is true also , to a lower extent in twins .

- SEASON :

It is more marked in winter due to deficient and lower exposure to sunlight . It reaches its peak in March and then declines in late June and July .

In temporate zone , it is more prevalent among coloured , dark skinned than white children , as the pigment of skin protects against the effect of sunlight , so prevents the penetration of ultraviolet rays . (MACKAY , 1975). The disease mostly occurs more in urban areas than in rural areas due to sunlight which is more in rural areas , due to open life . (HARRISON ,1966). In tropical countries the incidence decreased due to the presence of a lot of sunshine . Children living in sub-tropical and northern climates suffer still from the problem .

- EPIDEMIOLOGICAL FACTORS:

1- Bad housing condition .

2- Prolonged feeding on breast milk , also feeding on animal milk without vitamin "D" supplement or exposure to sunlight , as both human and animal milk , contain little amounts of vitamin "D".

3- Low socio-economic condition with defective diet .

4- Repeated attacks of diarrhea , gastroenteritis which affect children frequently in developing countries .(MAXCY, 1980) .

MATERIAL AND METHOD

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This study was performed on fifty infants and children of both sexes , from (6 months to 2 years of age) . Half of this group were rachitic children , while the others were healthy and were examind as a control group .

This group was taken from Zagazig Maternal & Child Health Center & Hospital in 1984 . These children attended the clinic for the treatment & vaccination .

The mothers of these children were interviewed ∝ their information was recorded in special files . Each file contained several records as shown in figure (1) .

FIGURE (I):

STUDIES ON RICKETS & ITS EFFECT ON
HEALTH AND GROWTH OF PRE-SCHOOL CHILDREN

I- MATERNITY & OBESTETRIC RECORDS:

- Mother's name .....
- Age .....

Residence .....

Occupation .....

Mother's feeding during pregnancy & Labour.....

Exposure to diseases during pregnancy .....

Labours : .. .normal.....or oeserian.....

: Date:... early.....or late.....

Mother 's feeding during lactation .....

II- CHILD'S RECORDS:

Name.....

Age.....

FEEDING.....BREAST.....OR ARTIFICIAL.....

Weaning.....early.....or delayed.....

Type of feeding.....

Exposure to sunlight .....

Child's disease :

-Nutritional.....

-Infectious.....

-Others.....

Growth :

-Normal or delayed .

- Teething .
- Walking .
- Talking .
- Others .

CLINICAL EXAMINATION :

- child's weight & height.....
Head & chest circumference .....
- Anterior & posterior fontanelle closure .....
- Teething examination .....
- Bowing leg .....
- Rachitic rosary .....
- Harison 's sulcus .....

RESULTS

RESULTS

TABLE (1) :

DISTIBUATION OF THE GROUP ON THE AREA OF SHARKIA WHERE THEY LIVE ,(CITY OR RURAL - AREA) .

THE AREA OF LIVING	RACHITIC		NON RACHITIC	
	NO.	%	NO.	%
IN ZAGAZIG CITY	5	20	15	60
IN RURAL AREA	20	80	10	40
TOTAL	25	100	25	100

This table shows that (80 %) of rachitic children were resident in the rural area , compared to (40 %) of non rachitic group .While those who were resident the urban area ,(ZAGAZIG CITY) , the rachitic group were less than the non rachitic (20%) and (60 %) .

By chi square analysis the result is highly significant .

$X^2 = 8.4$ $P \leq 0.1$

This indicates that rickets is greatly affected by the area of living (rural or urban) , among the rachitic and non rachitic children .

N.B : Chi square is one of the test of significance in statistic . (EL. SHABRAWY, .1979).

TABLE (2) :

THE AGE DISTRIBUATION AMONG RACHITIC AND NON RACHITIC GROUPS .

AGE IN MONTHS	RACHITIC		NON RACTIC	
	NO.	%	NO.	%
6 - 11	17	68	11	44
12-17	5	20	4	16
18 +	3	12	10	40
TOTAL	25	100	25	100

In this table it is found that more than half of rachitic cases (68 %) are under one year of age and (88 %) are under 1 1/2 years of age .

This finding indicates that the incidence rate of rickets occurs in the 2nd half of the first year and the 1 st half of the second year of the child's life . This result is statistically significant .

$X^2 = 5.0$ $P/\_ 0.5$.

TABLE (3) :

BIRTH ORDER OF RACHITIC AND NON RACHITIC CHILDREN .

BIRTH ORDER	RACHITIC		NON RACHITIC	
	NO.	%	NO.	%
FIRST - SECOND	4	16	18	72
THIRD- FOURTH	15	60	67	24
FIFTH +	6	24	1	4
TOTAL	25	100	25	100

This table shows that more than half of rachitic children (60 %) are the third and fourth member of the family children . While (16 %) of them are first and second in birth order and (24 %) are the fifth or more in the birth order . This finding indicates that the percetage of distribution of rickets increases with birth order . On the other hand we find that in non rachitic group,about 72 % of them are the first

or second in birth order , while 24 % of them are the third and fourth and 4 % of them are the fifth or more in birth order .

This finding explains that mothers are anxious for the health of the first babies , hence bring them to the infant clinic in Maternal and Child Health Centers for health care and vaccination .

By chi square analysis the result is highly significant.

$X^2 = 32.2$ $P < 0.1$

TABLE (4) :

SEX DISTRIBUTION AMONG RACHITIC AND NON - RACHITIC CHILDREN .

SEX DISTRIBUTION MALES OR FEMALES	RACHITIC NO.	%	NON RACHITIC NO.	%
MALES	14	56	12	48
FEMALES	11	44	13	52
TOTAL	25	100	25	100

This table shows that more than half of the rachitic cases (56 %) are males compared to the non rachitic group (48 %) ,. On the other hand , the rachitic females are less than the non rachitic (44 % and 52 %) .

This indicates that rickets affects males much .

The result statistically is highly significant .

$X^2 = 15{\cdot}7$ $P < 0.1$

TABLE (5) :

THE TYPE OF FEEDING AMONG RACHITIC AND NON RACHITIC CHILDREN .

FEEDIG	RACHITIC		NON RACHITIC	
	NO.	%	NO.	%
BREAST FEEDING	20	80	19	76
ARTIFICIAL FEEDING .	4	16	4	16
MIXED	1	4	2	8
TOTAL	25	100	25	100

In this table , it is found that most children of both groups , (rachitic and non rachitic) were breast fed , (80 % and 76 %) , while the artificial feeding among rachitic and non rachitic children was (16 % and 16 %) and in the mixed feeding the non - rachitic were double the percentage of the rachitic children (8 % and 4 %) .

This indicates that breast feeding without vitamin D supply increases the incidence of rickets among infants and children in their early childhood .

This result is statstically insignificant , that type of feeding does not affect rickets so much . $X^2 = 0.6$

TABLE (6) :

TEETH DELAY AMONG THE RACHITIC CHILDREN.

TOOTH	DELAYED TEETHING	
	NO.	%
CENTRAL INCISORS	11	44
LATERAL INCISORS	16	64
CUSPID	14	56
FIRST MOLARS	7	28

The presence of teeth was looked for . All twenty five cases showed delay in dentition . Table (6) , indicates the number and percentage of children with delayed dentition for each tooth in relation to specified age in months at which it should appear .

Almost (44 %) of the cases showed delayed dentition for the central incisors which should erupt between 6 to 8 months . The situation was even more worse

when we looked at the data for the lateral incisors delay .It is found that more than half of the cases (64 %) were with delayed lateral incisors eruption . The appearance of cuspids was delayed in (56 %) of cases , and for the first molars the percentage was 28 % .

It is clear from the result that delayed dentition for teeth eruption between 6 - 11 months (central and lateral incisors) , involved the majority of rachitic children .

TABLE (7) :

DELAYED MOVEMENT ACTIVITY AMONG RACHITIC AND NON RACHITIC CHILDREN .

MOVEMENT ACTIVITY	AGE IN MONTHS	DELAY IN ACTIVITY	
		NO.	%
SITTING ALONE	6M	2	8
STANDING ALONE	8 M	2	8
CRAWLING	10 M	14	56
WALKING ALONE	12 M	16	64
STANDING AND WALKING	15 M	7	28
CLIMBING STAIRS & RUNNING	18 M	7	28

Study of movement activity of the children in rachitic cases was done . The number and percentage of children with delayed movement activity are shown in table (7) .

Almost 8 % of patients showed delay in sitting alone at the age of 6 months . The delay in standing alone was also 8 % at the age of 8 months , and more than half of the cases (56 %) showed delay in crawling at the age of 10 months , while 64 % of cases could not

walk alone by the age of 12 months .

The activity of standing , walking , and climbing stairs showed delay in 14 cases i.e 56 % by the age of 15 months. These data showed that the percentage of children with delayed movements and activity , in relation to specified age at which it should occur , was greater during the second half of the first year compared to the first half of the second year .

TABLE (8) :

BONE DEFORMITIES AMONG THE RACHITIC CHILDREN .

BONE DEFORMITIES	NO.	%
DELAYED CLOSURE OF ANT. FONTANELLE	23	92
CRANIOTABES	18	72
HEAD LIKE BOX	9	63

TABLE (9) :

BONE DEFORMITY	NO.	%
LEG DEFORMITY	14	56
HAND DEFORMITY	20	80
RACHITIC ROSARY	14	56
KYPHOSIS	11	44

Bone deformities were examined and the data are presented in table (8) & (9) .

In table (8) , it is found that most cases (92 %) showed delayed closure of anterior fontanelle and 72 % of cases were with craniotabes . These data were presented in relation to specified age in months at which the closure of anterior fontanelle and craniotabes can occur .And 36 % of the cases were with head like box .

On the other hand , table (9) , showed that 80 % of cases were with broadened wrist (hand deformity) , and 56 % of cases were with leg deformity and also, 56 % of cases were with rachitic rosary , while 44 % of cases showed kyphosis .

DISCUSSION

DISCUSSION

Rickets is a systemic metabolic disease resulting from some sort of disturbances in calcium and phosphorus metabolism , characterized by defective mineralisation of bone , therefore it is considered as a nutritional problem related to many other aetiological and socio-economic factors causing undernutrition in Egypt . (NELSON,1975).

This study aims at determining the effect of rickets on the growth and health of infants and pre-school children in SHARKIA GOVERNORATE . It is performed on fifty infants and children of both sexes , at the age of (6 months to 2 years) , who attend the Maternal and Child Health Center and Hospital in 1984 .

We chose half of these children as a control group who were healthy and attending the clinic for vaccination , while the the other half were rachitic children .
The mothers of these children were interviewed and their information was recorded .

By analysis of this information , the results revealed that the presence of rickets affecting infants and children

depends on many factors which are :

1- Urbinization .

2- Age .

3- Birth order .

4- Sex .

5- Type of feeding . (breast or artifical) , without vitamin D supplement and exposure to ultraviolet rays of the sun .

These factors are supported by NELSON,I375, and ELLIES,1968.

Also, considerable attention has been given to the effect of rickets on the growth and development of infants and children in their first two years of life . The result showed that rickets can affect :

1- Dentition .

2- Movement activity .

3- Bone development .

On the other hand rickets may be associated with other nutritional diseases as , Protein-Caloric malnutrition (P.C.M),iron deficiency anaemia and also, with most of infectious diseases as bronchitis ,and gastroenteritis . (ELLIES, 1968).

From the result we find that :

1- Urbinization , age , birth order and sex are four significant factors affecting the spreading of rickets among infants and children .

By chi square analysis the results are significant ($P \leq 0.05$) .

2- Rickets can delay dentition , movement activity and leads to bone deformities . This delay is in accord to NELSON ,1975 and ELLIES ,1968 .

3- It was found that rickets in most cases may be associated with protein caloric malnutrition , iron deficiency anaemia gastroenteritis and bronchitis .

I Factors affecting the spreading of rickets among infants and children in the first two years of life are :

1- Urbinization

Present study showed that the rate of rickets was higher in rural area (80 %) than in urban area (20 %) of SHARKIA GOVERNORATE , This is compared to the non rachitic group of rural area (40 %)., This result is statistically significant ($P \leq 0.05$) . (table 1) .

HARRISON,1966, contradicted this result since he stated that rickets was more in urban areas than in rural areas .

But in our study the result may be due to the ignorance of mothers in rural area to supply their children with vitamin D, in adequate doses (400 IU) as a prophylaxis against rickets and also, due to their negligence in exposing them to sunlight. This result is in accord to FORFAR 1978 ,and DAVIDSON,1969.

2- Age :

It was found that age among rachitic and non rachitic children was statistically a significant factor since in table 2 , most rachitic children were at the age of (6 - 18 months of life) . This result is coincident with (ELLIES , 1968 & AEGERTER , 1975), who reported that rickets occurs more in the second half of the first year and the first half of the second year of life . Therefore we must pay more attention to the infants and children in this age by giving them prophylactic doses of vitamin D (400 IU / day). (CODEX , 1979) , and also, by their exposure to ultraviolet rays of the sun . (FORFAR , 1978) .

3 - BIRTH ORDER :

Table (3) , shows that most of the rachitic cases (60 %) are the third and fourth child in birth order of the family . This result indicates that the percentage of rickets increases according to birth order . This result is corrosponding with (SHUKRY , 1973), who reported that rickets increases with increasing the serial order of the child .

On the other hand we found that most of the non rachitic group (72 % of them) , were in the first and second child in birth order which explain that mothers are anxious for the health of the first babies , hence bring them to the infant clinic in Maternal and

Child Health Center . This is also due to the weakness of mothers' health after giving birth to a number of children .

4- SEX DISTRIBUTION AMONG RACHITIC AND NON RACHITIC CHILDREN:

It was found that more than half of the rachitic cases (56 %) were males , compared to the non rachitic group (48 %) . This indicates that rickets affects males more than females . By chi square analysis the result is significant .

As in our country people prefer males to females , I suppose that mothers keep them indoors and wrap them with much cloth , thus they deprive them from the exposure to ultraviolet rays of the sun .

5- TYPE OF FEEDING :

The present study indicates the importance of feeding as an essential factor in affecting children during their first two years of life since , in table (5) , most children of both groups (rachitic & non rachitic) were breast fed (80 % and 76 %), while lower incidence of rickets occured in artificial and mixed feeding . This result is coincident with (FORFAR, 1978)& BARNETT,1968) , who reported that breast milk is deficient

in vitamin "D" , since it contains about(0 to 10 U / 100 ml) of vit . D . Therefore it is recommended to supply infants & children with fortified milk with vit. D (400 IU / day) as a prophylxsis against rickets and expose them to ultraviolet rays . (DAVIDSON, 1969) .

ii THE EFFECT OF RICKETS ON GROWTH & HEALTH STATUS OF INFANTS & CHILDREN :

1- DENTITION :

It is clear that rickets delay dentition during the first years of the infant's life . This fact is supported by (ELLIES, 1968 & HARRISON, 1966).

In this study , the presence or absence of teeth was looked for., It was found that all twenty five cases showed delay in dentition while the control group showed no noticeable delay .

The results of dentition , indicated the number and percentage of children with delayed dentition for each tooth , in relation to specified age in which it should appear . (table 6) .

Almost 44% of cases showed delayed dentition for the central incisors which should erupt between 6-8 months .

The situation was even worse when we looked at the data for the lateral incisors delay . It was found that , more than

half of the rachitic cases (64 %) , showed delay in lateral incisors eruption . The appearance of cuspid were delayed in (56 %) of cases and for the first molar , the percentage was (28%) . It is clear from the result that delayed dentition for each teeth eruption between 6 - 11 months (central & lateral incisors) involved a majority of rachitic children. This result comes in accord to (ELLIES , 1968 & HARRISON , 1966), who stated that rickets delays dentition ,and as soon as vitamin "D" is given , the eruption of teeth appear rapidly (BARNETT, 1968) .

2- MOVEMENT ACTIVITY :

Study of movement activity of rachitic children was done. The number & percentage of delayed movement activity is shown in table (7) .

Rickets seems to delay particularly movement activity ; sitting, standing, and walking . (ELLIES , 1968 & WILSON , 1975) .

The present study indicates that , almost (8%) of the patients showed delay in sitting alone , at the age of 6 months . The delay in standing alone was (8%) at the age of 8 months , and more than half the cases (56%) showed delay in crawling at the age of 10 months, while (64%) of the cases could not walk alone by the age of 12 months . The activities of standing , walking , and climbing

stairs showed delay in 14 cases , i.e (56%) by age of 15 months. This indicates that the percentage of children with delayed movement activity in relation to specified age at which it should occur, was greater during the second half of the first year compared to the first half of the second year . This finding is corrosponding to the fact stated by (NELSON ,1975), who reported that rickets affects bone mineralisation and development of muscles &lacking in its tone which leads to delay in standing and walking .

3- BONE DEFORMITIES AMONG RACHITIC CHILDREN :

In table (8) , it is found that most cases (92%)showed delayed closure of anterior fontanelle , and (72%) of cases were with craniotabes ., and (36%) were with a head like box .
This data was presented in relation to specified age in months at which the closure of anterior fontanelle happens,since it completes its closure at the age of 15 months ,while craniotabes disappears at the end of the first year of the infant's life .(SLOBODY,1968)., on the other hand, table (9), showed that 80% of cases were with broad wrists (hand deformities), and (56%° of cases were with bowing legs and also (56%) of cases showed rachitic rosary, while (44%) suffered from kyphosis .
This result is corrosponding with (WILSON ,1971 & SLOBODY ,1968), who reported that changes in the bones in rickets result in gross manifestation recognizable clinically in enlargement of the wrist knee , bowed legs ,beading ribs (rachitic rosary),and craniotabes.

111 THE EFFECT OF RICKETS ON THE HEALTH STATUS OF CHILDREN:

In this study the presence or absence of nutritional and infecious diseases were looked for , it was found that most rachitic cases (80%) showed chest infection (bronchitis), while (64%) were with gastroenteritis and with previous repeated enteriti and bronchitis .

On the other hand , (44%°) of cases showed hypotonia of the muscle of the extremities and the abdomen ,which were associated with abdominal distension .

Most of the cases were underweight corresponding to specified age , they were pale showing iron deficiency anaemia and most of them were with protein-caloric malnutrition and marsmus .

This result is in accord to (ELLIES , 1968 & NELSON , 1975) , who reported that rickets is concurrently associated with under-nutrition and repeated gastroenteritis and bronchitis .

SUMMARY & RECOMMENDATION

SUMMARY AND RECOMMENDATION

Rickets is a systemic metabolic disease resulting from some sort of disturbances in calcium and phosphorus metabolism, characterized pathologically by defective mineralisation of bone and clinically by various skeletal, muscular and neurological manifestation . According to this definition , rickets is considered as a nutritional problem related to many other aetiological and socio-economic factors causing undernutrition in Egypt . (KHALIL, 1970).

This study was performed on fifty infants and children of both sexes , at the age of (6 months to two years) .
Half of them were rachitic children , while the other half were healthy and were considered as a control .
This group was taken from Zagazig Maternal and Child Health Center and Hospital in 1984 . The infants' mothers were interviewed for the history of the disease .

The results of this study revealed the following :

1- The highest percentage of rickets occured during the first year of life (68 %) , with an increase in the number of male children than the females (56 % & 44 %) .
2- There is a high prevalence of rickets among infants and children attending rural area (80%) than urban area(20 %).

3- The diatery history among rachitic and non rachitic cases showed that the majority of both groups were breast fed.

4- Infants receiving supplement of vitamin D are significally less prone to develop rickets .

5- A child is prone to develop rickets , if its serial number is high .

6- Rickets is concurrently associated with undernutrition and repeated gastroenteritis and bronchitis .

So, this study shows that rickets is a nutritional problem which faces our children in early childhood .
The aetiology in most cases was inadequate diatery intake of vitamin D and lack of exposure to ultraviolet rays .

Finally , we recommend that :

- Rickets should be paid more attention by health authorities .
- Prevention and treatment should start early .
- Supply of vitamin D, to pregnant women and lactating mothers should be given regularly in Maternal & Child Health Centers.
- Also, the mothers should be instructed to avoid excessive wrapping of their children and to expose them to sunlight frequently .
- Pregnant women , lactating mothers and children should be supplied with fortitied milk with vitamin D.

- The cases showing delayed dentition and movement activity should be considered as cases of pre-clinical rickets and should be given vitamin D , by intramuscular injection as a treatment . The dose to be given is 600,000 IU ,(FORFAR, 1978) , it should be repeated monthly for three successive doses .

- Twins and pre-mature babies should be paid more attention as they need bigger doses of vitamin D , in early months of life .

- Vitamin D resistant rachitic cases should be referred for investigation before increasing the doses of vitamin D .

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ARABIC SUMMARY

((الملخص باللغة العربيـة))

يعتبر مرض لين العظام فى الاطفال احد امراض سوء التغذية الذى يتميز باختلال فى تمثيل الكالسيوم والفوسفور فى الجسم مما ينتج عنه اختلال فى تكوين ونمو العظام والعضلات والذى يؤدى بدوره الى ظهور تشوهات فى عظام الاطراف وتأخر فى النمو والمشى والتسنين .

ومن ناحية أخرى ، فان هذا المرض يعتبر مشكلة غذائية تتعلق بكثير من العوامـــل البيئية والاجتماعية والاقتصادية فى مصر .

وان هذا البحث قد تم بدراسة ٥٠ طفل من الجنسين (بنين وبنات) فى السـن مابين ٦ شهور الى سنتين ـ وقد اعتبر نصف هذه الاطفال (٢٥ طفل) كمجموعة مقارنـة من الاطفال الاصحاء الذين تردد وا على مراكز رعاية الامومة والطفولة بالزقازيق للتطعـيم ضد الامراض ، كما اعتبر النصف الاخر (٢٥ حالة) من المصابون بمرض لين العظـام والذين تردد وا على المستشفى للعلاج فى عام ١٩٨٤ وقد اخذت بيانات ومعلومـات تتعلق بتاريخ المرض من امهات هؤلاء الاطفال وقد اعربت النتائج على التالى :ـ

١) ان اعلى نسبة للاصابة بمرض لين العظام حدثت فى العام الاول من حياة الطفل بنسبة ٦٨٪ مع زيادة نسبة اصابة البنين عن البنات بنسبة (٥٦٪ بنين و ٤٤٪ للبنات) .

٢) ان الاصابة بهذا المرض تنتشر فى الريف عنه فى المدينة بنسبة ٨٠٪ فى المناطـق الريفية و ٢٠٪ فى المدن .

٣) يزداد انتشار هذا المرض بزيادة ترتيب الطفل بين اطفال الاسرة .

٤) ان الاطفال الذين يتناولون كميات كافية من فيتامين د اقل عرضة للاصابة بمرض لين العظام عن الذين لا يتناولونه .

٥) وجد ان هناك علاقة وثيقة بين مرض لين العظام ووجود النزلات المعوية والشعبية في الاطفال حيث تزيد نسبة الاصابة بهذا المرض في اولئك الاطفال المصابون بهذه الامراض .

لذلك فقد وجد من الدراسة ان مرض لين العظام يعتبر احد امراض سوء التغذية التي تواجه الاطفال في السنوات الاولى من عمر الطفل ، والذي يتسبب نتيجة لنقص فيتامين د في غذاء الطفل وكذلك نتيجة لقلة تعرض الطفل لاشعة الشمس اللازمة لتحويل فيتامين د تحت الجلد الى فيتامين فعال يمكن الاستفادة منه .

واخيرا فان نتيجة هذه الدراسة تجعلنا ننادى باتباع الملاحظات الاتية :

* ان مرض لين العظام يجب ان يحظى برعاية كبيرة من الاطباء المختصين في هذا المجال .
* يجب اتباع وسائل الوقاية والعلاج سريعا للقضاء على هذا المرض .
* يجب امداد السيدات الحوامل واللاتى يرضعن اطفالهن وكذلك اطفالهن بجرعات كافية من فيتامين د - وذلك عن طريق التردد بانتظام على مراكز رعاية الامومة والطفولة .
* يجب تعليم الامهات الوسائل السليمة في رعاية وتغذية اطفالهن ومنعهن من حجب اطفالهن عن التعرض لاشعة الشمس اللازمة لهم .

* يجب امداد الامهات الغير مقتدرة على الانفاق بكميات كافية من الالبان المـــزودة بفيتامين د مجانا .
* يجب اعتبار الحالات التى تظهر اعراض اولية لوجود مرض لين العظام كحالة مرضيـــة تحتاج للعلاج باعطائها الجراعات المناسبة من فيتامين "د" (٦٠٠ر٠٠٠ وحدة) فى العضل مقسمة على ٣ جرعات مرة كل شهر . (فورفار ١٩٧٨) .
* الاطفال التوائم والضعفاء يجب اعطائهم اهتمام خاص وذلك بامدادهم بجرعات اكبر من (فيتامين "د") فى الشهور الاولى من حياتهم .
* حالات لين العظام التى تقاوم العلاج بفيتامين "د" يجب اكتشافها مبكرا حـــتى تتفادى مضاعفات زيادة هذا الفيتامين .

" بسم الله الرحمن الرحيم "

جامعة عين شمس
معهد الدراسات العليا للطفولة
قسم الدراسات الطبية والطب
نفسية

١٧

دراسة عن

" مرض لين العظام وتأثيره على الحالة الصحية والنمو لدى الاطفال فى سن ماقبل المدرسة "

رسالة مقدمة من

الطبيبة / فادية السيد الصباغ
بكالوريوس الطب والجراحة جامعة
عين شمس

توطئة للحصول على درجة الماجستير
فى دراسات الطفولة

تحت اشراف

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١٩٨٤