Mothers’ Knowledge Regarding vitamin A deficiency in under five children in Pediatric Teaching Hospital Wad Madani, Gezira State Sudan (2015).

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ADissertation

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Mothers’ Knowledge Regarding vitamin A deficiency in under five children in Pediatric Teaching Hospital Wad Madani, Gezira State Sudan (2015).

Asma Fadolalmola Ahmed Mohamed

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Mothers’ Knowledge Regarding vitamin A deficiency in under five children in Pediatric Teaching Hospital Wad Madani, Gezira State Sudan (2015).

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*Date: April/2016*
I dedicate this effort to my beloved parents, my dear husband for his unlimited support, my beloved kids who filled my life with joy and pleasure, to my sisters and brothers for their valuable support and encouragement, finally I dedicate this effort to my sincere friends.

Asma
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First and foremost I would like to thank my Almighty Allah for giving me strength and health to completed this work. I sincerely thanks all those who helped me to carry out this study.

University of Gezira, faculty of applied medical science.

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Mothers' Knowledge Regarding vitamin A deficiency in under five children in Pediatric Teaching Hospital Wad Madani, Gezira State Sudan (2015).

Abstract

Vitamin A deficiency is common in many developing countries, According to the World Health Organization, 190 million preschool-aged children around the world have serum retinol concentration below 0.70 micromoles/L. A descriptive hospital based study was conducted in Pediatric Teaching Hospital Wad Madani, aimed at assessing Mothers’ Knowledge Regarding vitamin A deficiency in under five children in Pediatric Teaching Hospital Wad Madani, Gezira State Sudan (2015). The study sample consisted of (70) women who attending with children in the period (from October to December -2015). Data was collected by using a questionnaire design for the study. Data analysis by using Statistical package for Social Sciences (SPSS). The study revealed that (40%) of the study sample illiterate and (61%) from study sample housewives. (58%) of study sample responded with correct answers regarding source of vitamin A, and (64.3%) of them responded correctly regarding their family like animal and plant resources rich vitamin A. and (67%) of the study sample responded with correct answers regarding their dealing when children refuse food rich in vitamin A, and (72.9%) of them responded correctly regarding symptoms of growth retardation. (87%) of study sample responded with correct answers regarding benefits of vitamin A. (88.6%) of them responded correctly regarding disease cause vitamin A deficiency. The study concluded that mothers knowledge regarding vitamin A deficiency were in adequate. The study recommended that educational program for mothers regarding Vitamin A deficiency must be done and design logbook and must be available in the hospitals.
العلماء يجاه نقص فيتامين (أ) عند الأطفال أقل من خمس سنوات بمستشفى الأطفال التعليمي بود مدني، ولاية الجزيرة السودان للعام 2015م

الاسماء: فضيلة المولى احمد

ملخص الدراسة

نقص فيتامين (أ) شائع في العديد من البلدان النامية، وعلي حسب منظمة الصحة العالمية، أن مائة وتسعة مليون من الأطفال في سن الدراسة حول العالم مصابين بنقص فيتامين (أ). أجريت هذه الدراسة الوصفية بمستشفى الأطفال التعليمي بود مدني، وهدفت إلى تقييم معرفة الأمهات تجاه نقص فيتامين (أ) لدى الأطفال دون سن الخامسة بمستشفى الأطفال التعليمي بود مدني، ولاية الجزيرة السودان عام 2015م. تكونت حجم عينة الدراسة من 70 أمهات البالغات حضرن بمستشفى الأطفال في الفترة من (أكتوبر إلى ديسمبر 2015م). جمع البيانات باستخدام استمارة استبيان تم تصميمها للدراسة. تم تحليل البيانات باستخدام برنامج الحزم الإحصائية للعلوم الاجتماعية "SPSS". أظهرت الدراسة أن 40% من أفراد العينة أميات و61% منهن ربات منزل. و58% من أفراد العينة كانت إجاباتهن صحيحة عن معرفة الأمهات بمصادر فيتامين (أ). 64% منهن كانت أجابتهن صحيحة عن معرفتهن بالمصادر الحيوانية والنباتية الغنية بفيتامين (أ). 67% من أفراد العينة أجابن بشكل صحيح عن كيفية التعامل مع أطفالهم الرافضين للطعام الغني بفيتامين (أ). 47% من أفراد العينة أجابوا على أعراض تأخر النمو و87% من أفراد أجابن بشكل صحيح معرفة فيتامين (أ). 88% من أفراد العينة أجابن بشكل صحيح على نقص فيتامين (أ) كانت غير كافية. أوصت الدراسة بعمل برامج تعليمية للأمهات عن نقص فيتامين (أ) وتوصيم كتبة تكون متاحة بالمستشفيات.
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Chapter One

Introduction
1. Introduction

1-1 Background:

Vitamin A deficiency is common in many developing countries. According to the World Health Organization, 190 million preschool-aged children and 19.1 million pregnant mothers around the world have a serum retinol concentration below 0.70 micromoles/L. In developing countries, low vitamin A intake is most strongly associated with health consequences during periods of high nutritional demand, such as during infancy, childhood, pregnancy, and lactation. In developing countries, vitamin A deficiency typically begins during infancy, when infants do not receive adequate supplies of colostrum or breast milk. Chronic diarrhea leads to excessive loss of vitamin A in young children, and vitamin A deficiency increases the risk of diarrhea. The most common symptom of vitamin A deficiency in young children and pregnant mothers is xerophthalmia. One of the early signs of xerophthalmia is night blindness, or the inability to see in low light or darkness (Amy V. Hass 2009).

Vitamin A deficiency is one of the top causes of preventable blindness in children. People with vitamin A deficiency (and, often, xerophthalmia with its characteristic Bitot’s spots) tend to have low iron status, which can lead to anemia. Vitamin A deficiency also increases the severity and mortality risk of infections (particularly diarrhea and measles) even before the onset of xerophthalmia. Deficiency of vitamin A is found among malnourished, elderly, and chronically sick populations in the United States, but it is more prevalent in developing countries. Abnormal visual adaptation to darkness, dry skin, dry hair, broken fingernails, and decreased resistance to infections are among the first signs of vitamin A deficiency. (Ball, George F. M. 2004)

1-2 Problem statement:

vitamin A deficiency is a lack of vitamin A in humans. It is common in poorer countries but rarely seen in more developed countries. Nyctalopia (night blindness) is one of the first signs of vitamin A deficiency. Xerophthalmia, keratomalacia, and complete blindness can also occur since Vitamin A has a major role in phototransduction. Vitamin A deficiency is the leading cause of preventable childhood blindness and is critical to achieving Millennium Development Goal 4:2 to reduce child mortality. [Dunne, 2012]. Approximately 250,000 to 500,000 malnourished
children in the developing world go blind each year from a deficiency of vitamin A, approximately half of whom die within a year of becoming blind. The United Nations Special Session on Children in 2002 set a goal of the elimination of vitamin A deficiency by 2010. The prevalence of night blindness due to vitamin A deficiency is also high among pregnant mothers in many developing countries. Vitamin A deficiency also contributes to maternal mortality and other poor outcomes in pregnancy and lactation. Vitamin A deficiency also diminishes the ability to fight infections. In countries where children are not immunized, infectious diseases like measles have higher fatality rates. As elucidated by Alfred Sommer, even mild, subclinical deficiency can also be a problem, as it may increase children's risk of developing respiratory and diarrheal infections, decrease growth rate, slow bone development, and decrease likelihood of survival from serious illness. [Briefec and Johns, 2007].

Vitamin A deficiency is estimated to affect approximately one third of children under the age of five around the world. It is estimated to claim the lives of 670,000 children under five annually. Approximately 250,000–500,000 children in developing countries become blind each year owing to vitamin A deficiency, with the highest prevalence in southeast Asia and Africa. According to the World Health Organization (WHO), vitamin A deficiency is under control in the United States, but, in developing countries, vitamin A deficiency is a significant concern. Globally, 69% of all children aged 6 to 59 months received two doses of vitamin A in 2011, fully protecting them against vitamin A deficiency (81% in the least developed countries). [Common Pregnancy Concerns” 2012].

Vitamin A deficiency can result from inadequate intake, fat malabsorption, or liver disorders. Deficiency impairs immunity and hematopoiesis and causes rashes and typical ocular effects (xerophthalmia, night blindness). Diagnosis is based on typical ocular findings and low vitamin A levels. Treatment consists of vitamin A given orally or, if symptoms are severe or malabsorption is the cause, parenterally. Vitamin A is found in many foods, including leafy green vegetables, orange vegetables (carrots, sweet potatoes, pumpkin, eggs, and cantaloupes). A lack of access to a balanced diet with enough vitamin A can lead to vitamin A deficiency. Vitamin A plays an important role in your vision. To see the full spectrum of light, your eye needs to produce certain pigments for the photoreceptor cells in your retina to work properly. Vitamin A deficiency stops the production of these pigments, leading to night
blindness. Your eye also needs vitamin A to nourish other parts of your eye, including the cornea, the clear covering on the front of your eye. Without enough vitamin A, your eyes cannot produce enough moisture to keep them properly lubricated. Vitamin A deficiency is the leading cause of preventable blindness in children worldwide. An estimated 250,000 to 500,000 children become blind every year because of vitamin A deficiency. Half of these children die within a year of losing their sight. In pregnant mothers, vitamin A deficiency causes night blindness and may contribute to maternal mortality. Vitamin A deficiency also compromises the immune system, measles and diarrhea [Common Pregnancy Concerns"2012].

1-3 justification:

Vitamin A deficiency is common in many developing countries, often because residents have limited access to foods containing preformed vitamin A from animal-based food sources and they do not commonly consume available foods containing beta-carotene due to poverty. According to the World Health Organization, 190 million preschool-aged children and 19.1 million pregnant mothers around the world have a serum retinol concentration below 0.70 micromoles/l. In these countries, low vitamin A intake is most strongly associated with health consequences during periods of high nutritional demand, such as during infancy, childhood, pregnancy, and lactation. In developing countries, vitamin A deficiency typically begins during infancy, when infants do not receive adequate supplies of colostrums or breast milk. Chronic diarrhea also leads to excessive loss of vitamin A in young children, and vitamin A deficiency increases the risk of diarrhea. The most common symptom of vitamin A deficiency in young children and pregnant mothers is xerophthalmia. One of the early signs of xerophthalmia is night blindness, or the inability to see in low light or darkness (Gropper, S; Smith, J 2009). Vitamin A deficiency is one of the top causes of preventable blindness in children. People with vitamin A deficiency (and, often, xerophthalmia with its characteristic Bitot's spots) tend to have low iron status, which can lead to anemia. Vitamin A deficiency also increases the severity and mortality risk of infections (particularly diarrhea and measles) even before the onset of xerophthalmia (Gropper, S; Smith, J 2009).

1-4 Objectives:
1-4-1 General objective:
To study Mothers' Knowledge regarding vitamin A deficiency in children under five years in Pediatric Teaching Hospital Wad Madani, Gezira State, Sudan, (2015).

1-4-2 Specific Objectives:

- To assess mother's knowledge regarding aspect in vitamin A deficiency such as definition, source, benefit, complications of vitamin A deficiency, prevention of complication of vitamin A deficiency and treatment of vitamin A deficiency.
Chapter Two

Literature Review
2-1 Introduction:

Vitamin A is the name of a group of fat-soluble retinoids, including retinol, retinal, and retinylesters. Vitamin A is involved in immune function, vision, reproduction, and cellular communication. Vitamin A is critical for vision as an essential component of a protein that absorbs light in the retinal receptors, and it supports the normal differentiation and functioning of the conjunctival membranes and cornea. Vitamin A supports cell growth and differentiation, playing a critical role in the normal formation and maintenance of the heart, lungs, kidneys, and other organs. Vitamin A deficiency is one of the top causes of preventable blindness in children (RASMUSEEN, 2009). People with vitamin A deficiency (and, often, xerophthalmia with its characteristic Bitot’s spots) tend to have low iron status, which can lead to anemia. Vitamin A deficiency increases the severity and mortality risk of infections (particularly diarrhea and measles) even before the onset of xerophthalmia. Vitamin A (retinol) is required for the formation of rhodopsin, a photoreceptor pigment in the retina. Vitamin A helps maintain epithelial tissues. Normally, the liver stores 80 to 90% of the body’s vitamin A. (European, 2008).

2-2 Definition of Vitamin A deficiency:

Vitamin A deficiency is a major public health nutrition problem in the developing world. It especially affects young children, among whom deficiency can cause xerophthalmia and lead to blindness, limit growth and increase the risk of death. It is also becoming clear that vitamin A deficiency can extend through school age and adolescent years into adulthood. Although the health consequences of vitamin A deficiency are not well delineated beyond early childhood, recent data indicate that vitamin A deficiency is a public health problem in Sudan. Vitamin A deficiency is estimated to affect approximately one third of children under the age of five around the world. It is estimated to claim the lives of 670,000 children under five annually. (WAGNER, 2012). Worldwide, vitamin A deficiency is estimated to affect approximately one third of children under the age of five around the world. It is estimated to claim the lives of 670,000 children under five annually. (PEARCE, 2007). In Sudan: there is evidence that vitamin A deficiency is a public health problem in the developing countries: approximately 250,000–500,000 children in developing countries become blind each year owing to vitamin A deficiency, with the highest prevalence in Southeast Asia and Africa. According to the World Health Organization (WHO), vitamin A deficiency is under control in the United States, but, in developing countries, vitamin A deficiency is a significant concern. Globally, 69% of all children aged 6 to 59 months received two doses of vitamin A in 2011, fully protecting them against vitamin A deficiency (81% in the least developed countries).
eastern sudan and among communities from western and southern sudan living around greater khartoum, famine conditions and civil unrest. there are reports indicative of vitamin a deficiency problem in the central and the far western provinces. there were no reports from the northern provinces. the need for a surveillance system was discussed. [Hibbard BM 2006].

**International:**
clinical and subclinical vitamin a deficiency are problems in at least 75 countriesin 1994, the who classified countries as having clinical or subclinical, severe, moderate, or mild vitamin a deficiency. clinical vitamin a deficiency (in which children demonstrate ophthalmic signs and symptoms, including blindness) occurs mainly in countries in southeast asia and sub-saharan africa.severe vitamin a deficiency is also found in persons in refugee settlements and in displaced populations[lavon,2007]

**United States:**
vitamin a deficiency is uncommon in the general population, but subgroups of patients suffering from fat malabsorption, cholestasis, or ibd or who have undergone small-bowel bypass may have subclinical deficiency with dark-adaptation abnormalities in the range of 60%. vegans, persons with alcoholism, toddlers and preschool children living below the poverty line, and recent immigrants or refugees from developing countries all have increased risk of vitamin a deficiency secondary to decreased ingestion.[horrocks,2011].

**Developing countries:**
An estimated 250 million children are at risk for vitamin deficiency syndromes. The most widely affected group includes up to 10 million malnourished children, who develop xerophthalmia and have an increased risk of complications and death from measles. Each year, 250,000-500,000 children become blind because of vitamin a deficiency. Improving the vitamin A status of children with deficiencies (aged 6-59 mo) can reduce measles and diarrhea mortality rates by 50% and 33%, respectively, and can decrease risk rates from all causes of mortality by 23%. Routine distribution of vitamin A to children in endemic areas leads to a decrease of childhood mortality .Vitamin A is an essential vitamin required for vision, gene transcription, boosting immune function, and great skin health. A deficiency in vitamin A can lead to blindness and increased viral infection, however deficiency is only considered a problem in developing countries where it is a leading cause of blindness in children.
Overconsumption of vitamin A can lead to jaundice, nausea, loss of appetite, irritability, vomiting, and even hair loss. Vitamin A is a fat soluble vitamin, and therefore, needs to be consumed with fat in order to have optimal absorption. High vitamin A foods include sweet potatoes, carrots, dark leafy greens, winter squashes, lettuce, dried apricots, cantaloupe, bell peppers, fish, liver, and tropical fruits. The current daily value for Vitamin A is 5000 international units (IU). [Lavon, 2007].

**In Sudan:**

This short review summarizes all the published and unpublished reports on vitamin A deficiency in the Sudan in the last four decades. Different local terms used by people to indicate vitamin A deficiency were enlisted. There is evidence that vitamin A deficiency is a public health problem in eastern Sudan and among communities from western and southern Sudan living around Greater Khartoum, who were displaced from their homelands because of drought, famine conditions and civil unrest. There are reports indicative of vitamin A deficiency problem in the central and the far western provinces. There were no reports from the northern provinces. The need for a surveillance system was discussed. [Davies, M. 2007].

**In Children**

Vitamin A deficiency affects about 190 million preschool-age children, mostly from Africa and South-East Asia. In infants and children, vitamin A is essential to support rapid growth and to help combat infections. Inadequate intakes of vitamin A may lead to vitamin A deficiency which can cause visual impairment in the form of night blindness and may increase the risk of illness and death from childhood infections, including measles and those causing diarrhoea. Vitamin A can be safely provided to children in a large dose, rather than more frequent smaller doses, as it can be stored by the body and released over time as needed. Many countries have successfully integrated strategies to deliver vitamin A supplements to infants and children in their national health policies, including delivery during routine health visits and immunizations. (Haider et al, 2012).

**2-3 Groups at Risk of Vitamin A Deficiency:**

The following groups are among those most likely to have inadequate intakes of vitamin A: Premature Infants, Infants and Young Children in Developing Countries. Most people with cystic fibrosis have pancreatic insufficiency, increasing their risk of vitamin A deficiency due to difficulty absorbing fat. Several cross-sectional studies
found that 15%–40% of patients with cystic fibrosis have vitamin A deficiency. However, improved pancreatic replacement treatments, better nutrition, and caloric supplements have helped most patients with cystic fibrosis become vitamin A sufficient. Several studies have shown that oral supplementation can correct low serum beta-carotene levels in people with cystic fibrosis, but no controlled studies have examined the effects of vitamin A supplementation on clinical outcomes in patients with cystic fibrosis (Young and Jewelly, 2007).

2-4Vitamin A Sources and Benefits:

Vitamin A is a fat-soluble vitamin that is good for healthy vision, skin, bones and other tissues in the body. Vitamin A often works as an antioxidant, fighting cell damage, but it also has many other uses. “Through its role with cell growth and division, vitamin A has an important role in the normal formation and maintenance of the heart, lungs, kidneys and other vital organs,” Dr. Sherry Ross, mothers’ health expert at Providence Saint John’s Health Center in Santa Monica, California, told Live Science. (Rasmussen KM 2009)

Sources

There are two types of vitamin A. Preformed vitamin A, also called retinol, is found in animal products. Good sources are fortified milk, eggs, meat, cheese, liver, halibut fish oil, cream and kidneys. Pro-vitamin A is found in plant-based foods such as fruits and vegetables, according to the United State National Library of Medicine (NLM). The most common type of pro-vitamin A is beta-carotene, a carotenoid that produces dark pigments in plant foods. Beta-carotene can be found in these brightly colored foods: Cantaloupe, Pink grapefruit, Apricot, Carrots, Pumpkin, Sweet potatoes, Winter squash, Dark green, leafy vegetables, Broccoli. (Rasmussen KM 2009)

Benefits:

Vitamin A has many varied functions. Retinol not only creates the pigments in the retina of the eye, according to NLM, but also is integral for good vision, especially night vision, and overall eye health. An age-related eye disease study by the National Eye Institute found that taking high levels of antioxidants, such as vitamin A, along with zinc, may reduce the risk of developing advanced age-related macular degeneration by about 25 percent. Age-related macular degeneration is the most common cause of loss of vision in the older population, said Ross. Vitamin A also helps skin grow and repair skin. “This being the case, it is the active ingredient in most
Retin-A type products out today,” said Dr. David Greuner, director and co-founder of NYC Surgical Associates. Retin-A is a brand name for tretinoin, a prescription medication that treats acne and other skin conditions. "It works by signaling to the cells to grow at a faster rate, bringing fresher, more youthful skin to the surface more rapidly. Used in excess, it can be quite irritating, however. (Rasmussen KM 2009).

Other functions of vitamin A include the formation and maintenance of teeth, bones, soft tissue, white blood cells, the immune system and mucus membranes. Beta-carotene also acts as an antioxidant, protecting cells from free radical damage. Though many antioxidants prevent cancer, there is no evidence that beta-carotene supplements are helpful in the prevention of cancer, according to the National Cancer Institute. On the other hand, all-natural beta-carotene that can be consumed through vegetables and fruits has been found to helpful in preventing cancer in many studies(Rasmussen KM 2009).

2-5 History of vitamin A deficiency:

Subclinical forms of vitamin a deficiency may not cause any symptoms, but the risk of developing respiratory and diarrheal infections is increased, the growth rate is decreased, and bone development is slowed. Patients may have a recent history of increased infections, infertility, development. The patient may also report increased fatigue, as a manifestation of vitamin a deficiency anemia (Haideretal,2012).

2-6 Etiology of vitamin A deficiency:

Primary vitamin A deficiency is usually caused

Prolonged dietary deprivationIt is endemic in areas such as southern and eastern Asia, where rice, devoid of β-carotene, is the staple food. Xerophthalmia due to primary deficiency is a common cause of blindness among young children in developing countries.[Murtoff and Heidi,2010]

Secondary vitamin A deficiency may be due to

- Decreased bioavailability of provitamin A carotenoids
- Interference with absorption, storage, or transport of vitamin AInterference with absorption or storage is likely in celiac disease, cystic fibrosis, pancreatic insufficiency, duodenal bypass, chronic diarrhea, bile duct obstruction, giardiasis, and cirrhosis. Vitamin A deficiency is common in prolonged protein-energy undernutrition not only because the diet is deficient but also because vitamin A storage and transport is defective. In children with complicated measles, vitamin A can shorten the duration of the disorder and reduce the severity of symptoms and risk of death(lauraetal,2012).
2-7 Vitamin A deficiency risks:

Vitamin A deficiency is a significant problem in developing nations in Africa and Southeast Asia. Young children and pregnant mothers in low-income countries are at highest risk. The main symptom of vitamin A deficiency is vision loss and blindness. Vision loss often begins as a problem adjusting to seeing in the dark, or night blindness. People with night blindness do not see well in the dark, but are able to see normally if enough light is present. As the vitamin A deficiency worsens, the conjunctiva, the covering on the white of the eye that helps lubricate your eye, dries out, and corneal ulcers appear. The progression of the deficiency eventually leads to vision loss and blindness. Vitamin A deficiency is diagnosed by an eye exam and by reviewing medical history. A blood test can measure the amount of vitamin A in the blood. However, because vitamin A deficiency is most common in areas with limited medical access, the diagnosis is often made on the basis of information provided by the parent about the child’s vision, particularly the appearance of night blindness (Laura Riley (2006)).

2-7-1 Vitamin A deficiency with pregnancy:

Vitamin A supplementation during pregnancy as part of routine antenatal care for the prevention of maternal and infant morbidity and mortality is not recommended

effect of Vitamin A Deficiency on the fetus:

Pregnant laboratory animals with severe vitamin A deficiency are less fertile, tend to resorb their fetuses or abort their pregnancies, and bear malformed offspring. Additionally, vitamin A is essential for the development of vital organ systems, particularly the lung. However there is very little evidence with which to judge the impact on fetal health of maternal vitamin A deficiency as commonly observed in human populations. Cases have been reported of congenital ocular defects in infants born to mothers who were very vitamin A deficient. It is plausible that these defects were caused by vitamin A deficiency, because of the similarity with animal studies. However, these mothers undoubtedly had many nutritional deficiencies and perhaps other illnesses that might also explain this finding. A question of greater public health importance is whether maternal vitamin A deficiency compromises the growth and vitamin A stores of the fetus. If maternal vitamin A deficiency contributes to low birth weight, maternal vitamin A deficiency would significantly impact infant mortality. Here again, animal studies modeling severe deficiency suggest the possibility, but
there is no direct evidence from human studies. Fetal weight gain and accumulation of vitamin A are strongly associated in animals and humans (that is, smaller fetuses have lower vitamin A stores), but it is not clear that vitamin A is limiting growth. Possibly, low weight gain and vitamin A gain result from general fetal malnutrition, due to inadequate placental size, function, or blood perfusion. Animal studies also suggest that fetal stores of vitamin A are even lower than normal when the mother is very deficient. However, because the vitamin A stores of newborns are so small, variation in the stores of newborns can have little direct impact on the subsequent vitamin A status of the breastfed infant. A few days intake of vitamin A from breast milk is equal to a healthy newborn. [Murtoff and Heidi, 2010]

2-7-2 Vitamin A Deficiency in children:

Vitamin A deficiency can result from inadequate intake, fat malabsorption, or liver disorders. Deficiency impairs immunity and hematopoiesis and causes rashes and typical ocular effects (xerophthalmia, night blindness). Diagnosis is based on typical ocular findings and low vitamin A levels. Treatment consists of vitamin A given orally or, if symptoms are severe or malabsorption is the cause, parenterally. Vitamin A deficiency is rare in the United States. However, vitamin A deficiency is common in many developing countries, often because residents have limited access to foods containing preformed vitamin A from animal-based food sources and they do not commonly consume available foods containing beta-carotene due to poverty. According to the World Health Organization, 190 million preschool-aged children and 19.1 million pregnant mothers around the world have a serum retinol concentration below 0.70 micromoles/L. In these countries, low vitamin A intake is most strongly associated with health consequences during periods of high nutritional demand, such as during infancy, childhood, pregnancy, and lactation. In developing countries, during infancy, when infants do not receive adequate supplies of colostrum or breast milk. Chronic diarrhea also leads to excessive loss of vitamin A in young children, A (Rasmussen KM, 2009).

The most common symptom of vitamin A deficiency in young children and pregnant mothers is xerophthalmia. One of the early signs of xerophthalmia is night blindness, or the inability to see in low light or darkness. Vitamin A deficiency is one of the top causes of preventable blindness in children. People with vitamin A deficiency (and, often, xerophthalmia with its characteristic Bitot’s spots) tend to have low iron status,
which can lead to anemia. Vitamin A deficiency also increases the severity and mortality risk of infections (particularly diarrhea and measles) even before the onset of xerophthalmia. Young children who are vitamin A deficient are at greater risk of morbidity and mortality than vitamin A–sufficient children. Diarrhea, respiratory infections, and measles are the diseases most frequently associated with a deficient vitamin A status. There are still some discrepancies in the results observed in various community trials: vitamin A supplementation does not always result in the expected decrease in morbidity, although mortality is usually reduced. Infants born to HIV-infected mothers are more vulnerable to disease, possibly, at least partly, because of the impairment of their immune system. [Murkoff, Heidi 2010]

2-8 Signs and symptoms of vitamin A deficiency:

Impaired dark adaptation of the eyes, which can lead to night blindness, is an early symptom. Xerophthalmia (which is nearly pathognomonic) results from keratinization of the eyes. It involves drying (xerosis) and thickening of the conjunctivae and corneas. Superficial foamy patches composed of epithelial debris and secretions on the exposed bulbar conjunctiva (Bitot spots) develop. In advanced deficiency, the cornea becomes hazy and can develop erosions, which can lead to its destruction (keratomalacia). Keratinization of the skin and of the mucous membranes in the respiratory, GI, and urinary tracts can occur. Drying, scaling, and follicular thickening of the skin and respiratory infections can result. Immunity is generally impaired. The younger the patient, the more severe are the effects of vitamin A deficiency. Growth retardation and infections are common among children. Mortality rate can exceed 50% in children with severe vitamin A deficiency [Rasmussen KM 2009].

2-9 Complications of vitamin A deficiency:

Night blindness:

is a condition making it difficult or impossible to see in relatively low light. It is a symptom of several eye diseases. Night blindness may exist from birth, or be caused by injury or malnutrition (for example, a lack of vitamin A). It can be described as insufficient adaptation to darkness. The most common cause of nyctalopia is retinitis pigmentosa, a disorder in which the rod cells in the retina gradually lose their ability to respond to the light. Patients suffering from this genetic condition have progressive nyctalopia and eventually their daytime vision may also be affected. In X-linked congenital stationary night blindness, from birth the rods either do not work at all, or
work very little, but the condition doesn't get worse. Another cause of night blindness is a deficiency of retinol, or vitamin A, found in fish oils, liver and dairy products (unne, Lavon J., ed. 2007).

The opposite problem, the inability to see in bright light, is known as hemeralopia and is much rarer. Since the outer area of the retina is made up of more rods than cones, loss of peripheral vision often results in night blindness. Individuals suffering from night blindness not only see poorly at night, but also require extra time for their eyes to adjust from brightly lit areas to dim ones. Contrast vision may also be greatly reduced. Rods contain a pigment called rhodopsin. When light falls on rhodopsin, it undergoes a series of conformational changes ultimately generating electrical signals which are carried to the brain via the optic nerve. In the absence of light, rhodopsin is regenerated. The body synthesizes rhodopsin from vitamin A, which is why a deficiency in vitamin A causes poor night vision.

- Vitamin A deficiency. Vitamin A is an essential vitamin that's found in carrots and yellow or green leafy vegetables. It helps keep the retina -- in the back of the eye -- healthy. Vitamin A deficiency is a rare cause of night blindness in the U.S. It occurs mostly in people with problems absorbing nutrients from the gastrointestinal tract. This might occur as a result of different diseases and conditions such as Crohn's disease, celiac disease, cystic fibrosis, or pancreatic insufficiency.

- Night blindness: Impaired vision in dim light and in the dark, due to impaired function of specific vision cells (namely, the rods) in the retina. The ability of our eyes to quickly view objects as they shift from light to dark areas and the ability to see in dim light or at night is an important part of our visual health. When we are not able to do such, the condition is referred to commonly as night blindness or medically as nyctalopia. It occurs as a result of various diseases that cause degeneration of the rods of the retina (the sensory cells responsible for vision in dim light). The problem can also appear as an inherited deficiency in visual purple, or rhodopsin, which is the pigment of the rods of the retina. The abnormality can also result from vitamin A deficiency. Rhodopsin, maintains its photosensitivity only in the presence of vitamin A.

- Night blindness is a classic finding from deficiency of vitamin A. It was described by the English physician William Heberden (1710-1801) who also discovered other medical disorders of importance including angina pectoris (chest pain that is often severe and crushing, due to an inadequate and nystanopia. supply of oxygen to the
heart muscle) and osteoarthritis of the small joints with nodules (Heberden's nodes) in and about the last joint of the finger (Leung et al., 2009).

- Sources of vitamin A include animal livers, milk, and yellow and green leafy vegetables which contain carotenes, chemically related substances that are converted to vitamin A in the body.
- Night blindness is also called day sight, nocturnal amblyopia, nyctalopia

The common cause of blindness in developing countries is vitamin A deficiency. The World Health Organization (WHO) estimates 13.8 million children to have some degree of visual loss related to vitamin A deficiency. Night blindness and its worsened condition, xerophthalmia, are markers of vitamin A deficiency, as it can also lead to impaired immune function, cancer, and birth defects. Collections of keratin in the conjunctiva pro-vitamin A precursors that will prevent vitamin A deficiency related night blindness. [WHO, 2011]

Night blindness is the difficulty for the eyes to adjust to dim light. Affected individuals are unable to distinguish images in low levels of illumination. People with night blindness have poor vision in the darkness, but see normally when adequate light is present. Vitamin A deficiency affects vision by inhibiting the production of rhodopsin, the eye pigment responsible for sensing low light situations. Rhodopsin is found in the retina and is composed of retinal (an active form of vitamin A) and opsin (a protein). Because the body cannot create retinal in sufficient amounts, a diet low in vitamin A will lead to a decreased amount of rhodopsin in the eye, as there is inadequate retinal to bind with opsin. Night blindness results [Williamson CS 2006]

Night blindness caused by vitamin A deficiency has been associated with the loss of goblet cells in the conjunctiva, a membrane covering the outer surface of the eye. Goblet cells are responsible for secretion of mucus, and their absence results in xerophthalmia, a condition where the eyes fail to produce tears. Dead epithelial and microbial cells accumulate on the conjunctiva and form debris that can lead to infection and possibly blindness. Decreasing night blindness requires the improvement of vitamin A status in at-risk populations. Supplements and fortification of food have been shown to be effective interventions. Supplement treatment for night blindness includes high doses of vitamin A (200,000 IU) in the form of retinyl palmitate to be taken by mouth, which administered two to four times a year. Intramuscular injections are poorly absorbed and are ineffective in delivering sufficient bio-available vitamin A. Fortification of food with vitamin A is costly, but can be done
in wheat, sugar, and milk.\textsuperscript{1} Households may circumvent expensive fortified food by altering dietary habits. Consumption of yellow-orange fruits and vegetables rich in carotenoids, specifically beta-carotene, provides (Williamson CS 2006).

**Xerophthalmia**

Xerophthalmia is the quintessential expression of vitamin A deficiency. Under conditions of gradually worsening vitamin A status, the eye undergoes a series of changes, beginning with night blindness (the inability to see under low levels of illumination). This reflects the essential role retinol plays in the formation of rhodopsin, the visual pigment essential to the retinal receptors responsible for dark adaptation [Lavon, 2007]. The Eber's Papyrus describes night blindness in ancient Egypt. Physicians treated the condition by squeezing the “juices” of a grilled lamb's liver into the eyes of afflicted patients. In 1971, George Wolff speculated that these topically applied “drops,” rich in retinol, probably drained into the lachrymal sac, where they were absorbed into the systemic circulation and thereby reached the retinal cells. Perhaps that was the case, but I observed the treatment of a young boy in rural Indonesia that was described in exactly the same fashion, but provided a more direct explanation for the way in which “liver juices,” applied topically, could reach the back of the eye. At the conclusion of the ceremony, after juice from a goat liver had been squeezed onto the boy's eyes, the traditional healer fed the child the remaining liver! The healer did not consider eating the liver part of the treatment; he fed the child the liver so as not to waste precious food [Rasmussen KM 2009]

**2-10 Infection rates of Vitamin A Deficiency:**

Along with poor diet there is a large amount of infection and disease present in many developing communities. Infection is very draining on vitamin A reserves and this vitamin A deficit leaves the individual more susceptible to infection (Combs, 2009). Increased of exporter xerophthalmia has been seen after an outbreak of measles and the varying stages of xerophthalmia become a good reference point for the extent of deficiency (with mortality increasing with severity of the eye disease). In a longitudinal study of preschool Indonesian children it was found that susceptibility to disease increased nine times when severe vitamin A deficiency was present (Dole 2009). The reason for the increased infection rate in vitamin A deficient populations is due to the T-killer cells which require retinoids to proliferate correctly (Athanassiades 2010). Retinoic acid binds the promoter region of specific genes and so
activates the transcription process and therefore cell replication (Baron 2011).

A vitamin A deficient diet will have a very limited surplus of retinol and so cell proliferation and replication will be suppressed, contributing to a reduced number of T-cells and lymphocytes. Suppression of these will result in a lack of immune reaction if pathogens become present in the body and consequently a greater susceptibility to incubation of disease. Vitamin A deficiency and infections aggravate each other and therefore with infection the vitamin A levels are depleted which in turn reduces intestinal absorption of vitamin A (WHO 2009). Very often seen with vitamin A deficiency is protein energy malnutrition (PEM). With PEM the synthesis of retinol binding protein (RBP) is decreased, consequently the uptake of retinol is reduced (Laura Riley 2006).

This leads to an inability to utilise any vitamin A present as the RBP is absent and so the retinol cannot be transported to the liver maximising the vitamin A deficiency (Hibbard BM 2006).

2-11 Diagnosis of vitamin A Deficiency:

Serum retinol levels, clinical evaluation, and response to vitamin A supplementation suggest the diagnosis. Dark adaptation can be impaired in other disorders (eg, zinc deficiency, retinitis pigmentosa, severe refractive errors, cataracts, diabetic retinopathy). If dark adaptation is impaired, rod scotometry and electroretinography are done to determine whether vitamin A deficiency is the cause. Serum levels of retinol are measured. Normal range is 28 to 86 μg/dL (1 to 3 µmol/L). However, levels decrease only after the deficiency is advanced because the liver contains large stores of vitamin A. Also, decreased levels may result from acute infection, which causes retinol-binding protein and transthyretin (also called prealbumin) levels to decrease transiently. A therapeutic trial of vitamin A may help confirm the diagnosis (lavon, 2007).

Today, vitamin A supplementation is the most efficient way of correcting vitamin A deficiency. Its only drawback is the potential risk of teratogenesis. Interesting attempts have been made to replace vitamin A with the provitamin β-carotene, which has never been associated with any teratogenic risk. When β-carotene was provided as a synthetic supplement, it was as efficient as vitamin A in reversing abnormal eye cytology, a clinical marker of vitamin A deficiency. Other authors found that the β-carotene of orange fruit (S de Pee, CE West, D Permaesih, S Maruti, Muhilal, JGAJ Hautvast, unpublished observations, 1997) was a more efficient source of vitamin A than dark-green leafy vegetables, probably because of the lower bioavailability of β-
carotene in the latter. Fostering the local production and utilization of sources of vitamin A is promising, yet the problem lies not only in the availability of vitamin A sources, but also in the economic status of the population. Such a distinction between areas where vitamin A deficiency is endemic (i.e., some low-income countries) and those where it is not (i.e., industrialized countries) is convenient, but may not reflect the real situation. It is quite possible, and in our opinion rather likely, that a significant portion of the low-income population in some of the most industrialized countries suffers from undiagnosed low vitamin A status. Mothers in these populations, who often do not undergo prenatal examinations, would benefit from a safely designed vitamin A supplementation protocol. However, to our knowledge, there has been no attempt to identify these mothers or to correct their nutritional deficiencies (Murkoff and Heidi, 2010).

2-12 Imaging Studies:

In children, radiographic films of the long bones may be useful when an evaluation is being made for bone growth and for excessive deposition of periosteal bone. The Procedures of Imaging Studies Dark-adaptation threshold should be tested in the United States, vitamin A deficiency can easily be prevented through the consumption of foods recommended in the Diet subsection. Treatment for subclinical vitamin A deficiency includes the consumption of vitamin A–rich foods, such as liver, beef, chicken, eggs, fortified milk, carrots, mangoes, sweet potatoes, and leafy green vegetables. The common cause of blindness in developing countries is vitamin A deficiency. The World Health Organization (WHO) estimates 13.8 million children to have some degree of visual loss related to vitamin A deficiency (WHO, 2010).

Night blindness and its worsened condition, xerophthalmia, are markers of vitamin A deficiency, as it can also lead to impaired immune function, cancer, and birth defects. Collections of keratin in the conjunctiva, known as Bitot's spots, are also seen. Imtiaz's sign is the earliest ocular sign of vitamin A deficiency. Conjunctival epithelial defects occur around lateral aspect of the limbus in subclinical stage of vitamin A deficiency. These conjunctival epithelial defects are not even visible on a biomicroscope, but they take up black stain and become readily visible after instillation of kajal (surma); this is called "Imtiaz's sign. Night blindness is the difficulty for the eyes to adjust to dim light. Affected individuals are unable to distinguish images in low levels of illumination. People with night blindness have poor vision in the darkness, but see normally when
adequate light is present. Vitamin A deficiency affects vision by inhibiting the production of rhodopsin, the eye pigment responsible for sensing low light situations. Rhodopsin is found in the retina and is composed of retinal (an active form of vitamin A) and opsin (a protein). Because the body cannot create retinal in sufficient amounts, a diet low in vitamin A will lead to a decreased amount of rhodopsin in the eye, as there is inadequate retinal to bind with opsin. Night blindness caused by vitamin A deficiency has been associated with the loss of goblet cells in the conjunctiva, a membrane covering the outer surface of the eye.

Goblet cells are responsible for secretion of mucus, and their absence results in xerophthalmia, a condition where the eyes fail to produce tears. Dead epithelial and microbial cells accumulate on the conjunctiva and form debris that can lead to infection and possibly blindness. Decreasing night blindness requires the improvement of vitamin A status in at-risk populations. Supplements and fortification of food have been shown to be effective interventions. Supplement treatment for night blindness includes high doses of vitamin A (200,000 IU) in the form of retinyl palmitate to be taken by mouth, which is administered two to four times a year. Intramuscular injections are poorly absorbed and are ineffective in delivering sufficient bio-available vitamin A. Fortification of food with vitamin A is costly, but can be done in wheat, sugar, and milk. Households may circumvent expensive fortified food by altering dietary habits. Consumption of yellow-orange fruits and vegetables rich in carotenoids, specifically beta-carotene, provides pro-vitamin A precursors that will prevent vitamin A deficiency related night blindness. (Lavon, 2007)

Impaired dark adaptation of the eyes, which can lead to night blindness, is an early symptom. Xerophthalmia (which is nearly pathognomonic) results from keratinization of the eyes. It involves drying (xerosis) and thickening of the conjunctivae and corneas. Superficial foamy patches composed of epithelial debris and secretions on the exposed bulbar conjunctiva (Bitot spots) develop. In advanced deficiency, the cornea becomes hazy and can develop erosions, which can lead to its destruction (keratomalacia). Keratinization of the skin and of the mucous membranes in the respiratory, GI, and urinary tracts can occur. Drying, scaling, and follicular thickening of the skin and respiratory infections can result. Immunity is generally impaired. The younger the patient, the more severe are the effects of vitamin A deficiency. Growth retardation and infections are common among children. Mortality rate can exceed 50% in children with severe vitamin A deficiency (Murkoff and Heidi, 2010).
2-13 Treatment of Vitamin A deficiency:

Vitamin A deficiency can be treated with vitamin A supplements. The amount of supplements depends upon the age of the child. Vitamin A supplements can reverse night blindness and help the eyes become properly lubricated again.

Treatment of vitamin a deficiency can be undertaken with both oral and injectable forms, generally as vitamin A palmitate. As an oral form, the supplementation of vitamin A is effective for lowering the risk of morbidity, especially from severe diarrhea, and reducing mortality from measles and all-cause mortality. Studies have shown that vitamin A supplementation of children under five who are at risk of vitamin a deficiency can reduce all-cause mortality by 23 per cent.[Williamson,2006]. Some countries where vitamin a deficiency is a public health problem address its elimination by including vitamin A supplements available in capsule form with National Immunization Days (NIDs) for polio eradication or measles. Additionally, the delivery of vitamin A supplements, during integrated child health events such as child health days, have helped ensure high coverage of vitamin A supplementation in a large number of least developed countries. Child health events enable many countries in West and Central Africa to achieve over 80 per cent coverage of vitamin A supplementation. According to UNICEF Data, in 2013 worldwide, 65 per cent of children between the ages of 6 and 59 months were fully protected with two high-dose vitamin A supplements. Vitamin A capsules cost about US$0.02. The capsules are easy to handle; they don’t need to be stored in a refrigerator or vaccine carrier. When the correct dosage is given, vitamin A is safe and has no negative effect on seroconversion rates for Oral Polio Vaccine or measles vaccine. However, because the benefit of vitamin A supplements is transient, children need them regularly every four to six months. Since NIDs provide only one dose per year, NIDs-linked vitamin A distribution must be complemented by other dose programs to maintain vitamin A in children[Murkoff and Heidi,2010].

Maternal high supplementation benefits both mother and breast-fed infant: high dose vitamin A supplementation of the lactating mother in the first month postpartum can provide the breast-fed infant with an appropriate amount of vitamin A through breast milk. However, high-dose supplementation of pregnant mothers should be avoided because it can cause miscarriage and birth defects.(murkoff and Heidi,2010).

- Food fortification is also useful for improving vitamin a deficiency. A variety of oily
and dry forms of the retinol esters, retinyl acetates and retinyl palmitate are available for food fortification of vitamin A. Margarine and oil are the ideal food vehicles for vitamin A fortification. They protect vitamin A from oxidation during storage and prompt absorption of vitamin A. Dietary diversification can also control vitamin A deficiency. Non-animal sources of vitamin A which contain pre-formed vitamin A account for greater than 80% of intake for most individuals in the developing world. The increase in consumption of vitamin A-rich foods of animal origin in addition to fruits and vegetables has beneficial effects on vitamin A deficiency. (Laura Riley 2006).

2-14 Prevention of vitamin A deficiency:

Prevention means to stop something from happening. Vitamin A supplementation completely “stops”/prevents childhood blindness from vitamin A deficiency. Invest in prevention and preventive interventions! Just 2 doses of vitamin A given annually to all children 6-59 months of age prevents their developing vitamin A deficiency blindness. Because vitamin A deficiency can have a range of consequences, including an increased risk of child mortality, the term now used is “vitamin A deficiency disorders” (vitamin a deficiencyd). The reason why there are global programmes for control of vitamin A deficiency in children is because it also significantly increases under 5 mortality rates in countries where it is a public health problem. Indeed, under 5 mortality rates are now used to indicate the likelihood that whether a country has significant vitamin A deficiency in its population of children. Improving a child’s vitamin A status enhances resistance to infections and reduces the overall risk of mortality by 24% (Murkoff, Heidi 2010).

A minimum coverage of 70% of children 6-59 months of age is required to observe reductions in childhood mortality (UNICEF, 2007).

The dosage recommended is one dose of vitamin A for children 6-11 months and two doses annually thereafter to the age of 59 months. The World Health Organization (WHO) recommends that all infants and children 6 - 59 months of age should receive vitamin A supplementation (VAS) if they reside in a country in which vitamin a deficiency is classified as being of either “moderate” or “severe” public health significance—this is known as Universal Supplementation. Universal Supplementation is currently recommended for 122 countries—73 countries with severe vitamin a deficiency and 49 countries with moderate vitamin a deficiency (WHO, 2010)

The diet should include dark green leafy vegetables, deep- or bright-colored fruits (egg, papayas, oranges), carrots, and yellow vegetables (egg, squash, pumpkin). vitamin A–fortified milk and cereals, liver, egg yolks, and fish liver oils are helpful.
Carotenoids are absorbed better when consumed with some dietary fat. If milk allergy is suspected in infants, they should be given adequate vitamin A in formula feedings. In developing countries, prophylactic supplements of vitamin A palmitate in oil 60,000 RAE (200,000 IU) po every 6 mo are advised for all children between 1 and 5 yr of age; infants < 6 mo can be given a one-time dose of 15,000 RAE (50,000 IU), and those aged 6 to 12 mo can be given a one-time dose of 30,000 RAE (100,000 IU) (Laura Riley 2006).

A review of the benefits and safety of high dose vitamin A for the treatment of common illnesses in children is helpful in identifying randomized, placebo-controlled trials. Only 24 out of almost 1,500 trials were designed adequately. There is substantial evidence that high dose vitamin A supplements reduce mortality from measles. In the Republic of South Africa418, and in Tanzania 419, providing 200,000 IU on two consecutive days significantly reduced morbidity and mortality in children hospitalized for measles, compared to a placebo group. A review of a large body of data from the Republic of South Africa revealed that high dose vitamin A, provided to children with measles reduced mortality to 1.6%, compared to 5% in untreated children420. It is now standard practice to provide high dose vitamin A during the treatment of measles. A placebo-controlled study, in South African children less severely affected with measles, showed no benefits of high dose vitamin A in reduction of respiratory symptoms(Hibbard,2006).

High dose vitamin A does not seem to be useful for the treatment of acute, watery diarrhea, according to three placebo-controlled trials: two in Bangladesh and one in India424. However, children with acute shigellosis in Bangladesh were cured more rapidly if given 200,000 IU of vitamin A425, and low dose (but not high dose) vitamin A significantly reduced the incidence of severe diarrhea in severely malnourished children in the D.R.Congo,(COCHRANE, 2011).

2-15 Consultations about vitamin A deficiency:
Consult endocrinologists, gastroenterologists, ophthalmologists, nutritionists, infectious disease specialists, and dermatologists, as indicated.

Diet:
The Dietary Guidelines for Americans, from the US departments of Agriculture Health Human Services, recommend consumption of a variety of foods for a comprehensive nutrient intake.1 Vitamin A – rich foods include the following: Liver, Beef, Chicken ,Eggs ,Whole milk, Fortified milk, Carrots, Mangoes, Orange fruits,
Sweet potatoes, Spinach, kale, and other green vegetables. Eating at least 5 servings of fruits and vegetables per day is recommended in order to provide a comprehensive distribution of carotenoids. A variety of foods, such as breakfast cereals, pastries, breads, crackers, and cereal grain bars, are often fortified with 10-15% of the RDA of vitamin A. The goals of pharmacotherapy are to reduce morbidity and to prevent complications. Along with poor diet there is a large amount of infection and disease present in many developing communities. Infection is very draining on vitamin A reserves and this vitamin A deficit leaves the individual more susceptible to infection increased documentation of xerophthalmia has been seen after an outbreak of measles and the varying stages of xerophthalmia become a good reference point for the extent of deficiency (with mortality increasing with severity of the eye disease) (strobleletal ,2007).

2-16 Nursing intervention:

2.16.1 Potential Nursing Diagnoses

- Nutrition, Imbalanced: Less than Body Requirements
- Sensory Perception, Disturbed, related to vitamin A deficiency
- Skin Integrity, Impaired
- Knowledge, Deficient, related to drug therapy

2.16.2 Assessment

- Obtain a complete health history including allergies, drug history, and possible drug interactions.
- Assess for the presence or history of vitamin A deficiency such as inadequate dietary intake, malabsorption diseases, and impaired liver function.
- Obtain baseline vision acuity examination.
- Assess integrity of skin and mucous membranes.
- Obtain serum vitamin A level, complete blood count (CBC), liver function profile, and serum protein and albumin levels.

2.16.3 Planning: Client Goals and Expected Outcomes

The client will:

- Exhibit improvement in serum vitamin A level.
- Immediately report side effects such as increased nausea, vomiting, headache, loss of hair, lethargy, and malaise.
- Demonstrate an understanding of the drug’s action by accurately describing drug side
2.16.4 Implementation

2.16.4.1 Client Education/Discharge

- A and oral contraceptives can cause toxic levels of vitamin A.
- Instruct client to:
  - Maintain a dietary log for 48 hours.
  - Eat foods rich in vitamin A such as egg yolks, butter, milk, liver, dark leafy vegetables, and orange fruits and vegetables.
  - Advise client to report any changes in vision.
  - Instruct client to:
    - Watch for signs and symptoms of vitamin A overdose such as nausea, vomiting, anorexia, dry skin and lips, headache, and loss of hair.
    - Immediately stop taking medication if signs of toxicity are noted.
    - Instruct client to:
      - Follow dosage directions given by the healthcare provider or on the label.
      - Immediately report any changes in neurologic status such as increased sleepiness, headaches, lethargy, and malaise.
      - Advise client to avoid laxatives that contain mineral oil.
      - Instruct client to:
        - Adhere to medication schedule and avoid double doses of the vitamin.
        - Keep scheduled laboratory visits for testing if taking oral contraceptives.

2.16.4.2 Interventions and (Rationales)

- Monitor client’s diet to determine intake of vitamin A foods. (Deficient state may be caused by poor dietary habits.)
- Periodically monitor visual acuity. (Vitamin A may cause miosis, papilledema, and nystagmus)
- Monitor for symptoms of vitamin A toxicity. (Storage of excess vitamin A can lead to hypervitaminosis)
- Monitor for signs of increased intracranial pressure. (Vitamin A may cause increased intracranial pressure if taken in large doses.)
- Monitor for drug interactions with oral contraceptives. (Concurrent use of vitamin
- Assess for use of mineral oil. (Mineral oil inhibits the absorption of vitamin A.)
2.16.5 Evaluation of Outcome Criteria

- Evaluate the effectiveness of drug therapy by confirming that client goals and expected outcomes have been met (see “Planning”).
- The client’s labs exhibit an improvement in vitamin A level.
- The client verbalizes side effects that require reporting.
- The client demonstrates an understanding of the drug’s action by accurately describing drug side effects and precautions. [briefec and johns, 2007]

2-17 Previous studies:

In a longitudinal study of preschool Indonesian children, it was found that susceptibility to disease increased nine times when severe vitamin A deficiency was present. The reason for the increased infection rate in vitamin A deficient populations is due to the T-killer cells which require retinoids to proliferate correctly. Retinoic acid binds the promoter region of specific genes and so activates the transcription process and therefore cell replication. A vitamin A deficient diet will have a very limited surplus of retinol and so cell proliferation and replication will be suppressed, contributing to a reduced number of T-cells and lymphocytes. Suppression of these will result in a lack of immune reaction if pathogens become present in the body and consequently a greater susceptibility to incubation of disease (Hibbard, 2006).

Vitamin A deficiency and infections aggravate each other, and therefore with infection the vitamin A levels are depleted, which in turn reduces intestinal absorption of vitamin A. Very often seen with vitamin A deficiency is protein energy malnutrition (PEM). With PEM, the synthesis of retinol binding protein (RBP) is decreased, consequently the uptake of retinol is reduced. This leads to an inability to utilise any vitamin A present as the RBP is absent and so the retinol cannot be transported to the liver, maximising the vitamin A deficiency (Georgieff, 2007).
Chapter Three

Materials and Methods
3-1 Study design:
   A descriptive hospital based study conducted at assessing mother’s knowledge regarding vitamin A deficiency in under five children during the period (November-December\ 2015).

3-2 Study area:
   The study was conducted in hospital for treatment and follow up in Wad Medani Pediatrics Teaching Hospital
   Wad Medani Pediatrics teaching hospital contain general department medicine (endocrine, nephrology, neurology, GIT and chest) (310 beds) (4) Theater, one referred clinic, one discharge clinic, and two dayS theater
   It’s composed of an emergency pediatric department, outpatient’s clinics, blood bank, laboratory, pharmacy and large neonatal care unit.

3-3 Study Population:
   Mothers in selected area and mother during the period of the study.

3-3-1 Inclusion criteria:
   Include all available mothers during the period of the study.

3-3-2 Exclusion criteria:
   Excluded mothers refused who attended during the period of the study.

3-4 Sample size:
   (70) Of Total covertly which was available during the period of the study.

3-5 Sample technique:
   The interviewer’s questionnaire method was used to obtain information on demographic data, and to assess the knowledge about vitamin A deficiency.

3-6 Data collection and analyzing:
   The data collected by using self administration questionnaire given to the participants. The data analyzed by using the SPSS program with statistical analysis and presented in tables and graphs.
Chapter four
Results and Discussion
4- Results and Discussion

4- 1 Results:

Table (4-1-1) : Distribution of the study sample according to their age and their educational level. (No.70)

<table>
<thead>
<tr>
<th>Item</th>
<th>No</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;20 years</td>
<td>8</td>
<td>11.4%</td>
</tr>
<tr>
<td>21-30 years</td>
<td>36</td>
<td>51.4%</td>
</tr>
<tr>
<td>&gt;40 years</td>
<td>26</td>
<td>37.2%</td>
</tr>
<tr>
<td>Total</td>
<td>70</td>
<td>100%</td>
</tr>
<tr>
<td>Educational level</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Illiterate</td>
<td>28</td>
<td>40%</td>
</tr>
<tr>
<td>Primary</td>
<td>18</td>
<td>25.7%</td>
</tr>
<tr>
<td>Secondary</td>
<td>14</td>
<td>20%</td>
</tr>
<tr>
<td>University</td>
<td>10</td>
<td>14.3%</td>
</tr>
<tr>
<td>Total</td>
<td>70</td>
<td>100%</td>
</tr>
</tbody>
</table>

Table (4-1-1) shows that (51.4%) of the study sample ages ranged between 20-30 years and (40%) of them at illiterate.
(4-1-2): Distribution of the study sample according to their occupation and monthly income (No.70)

<table>
<thead>
<tr>
<th>Item</th>
<th>NO</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Occupation</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Housewife</td>
<td>34</td>
<td>61.4%</td>
</tr>
<tr>
<td>Worker</td>
<td>15</td>
<td>21.4%</td>
</tr>
<tr>
<td>Student</td>
<td>12</td>
<td>17.2%</td>
</tr>
<tr>
<td>Total</td>
<td>70</td>
<td>100%</td>
</tr>
<tr>
<td>Monthly income</td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;500</td>
<td>15</td>
<td>21.4%</td>
</tr>
<tr>
<td>500-1000</td>
<td>13</td>
<td>18.6%</td>
</tr>
<tr>
<td>1500-1000</td>
<td>17</td>
<td>24.3%</td>
</tr>
<tr>
<td>Dialy income</td>
<td>25</td>
<td>35.7%</td>
</tr>
<tr>
<td>Total</td>
<td>70</td>
<td>100%</td>
</tr>
</tbody>
</table>

Table (4-1-2):
Results of table (2) shows that (61.4%) of the study sample housewife and (35.7%) at Daily income.
figure (1) Distribution of the study sample according to source of knowledge regarding vitamin A.

Figure (1) shows that (58.5%) of mothers sample their source of knowledge about vitamin A from television.
figure (2) Distribution of the study sample according to their responsibility about income in the family.

figure (2) shows that (68.6%) from sample responsible by fathers and (31.4%) from mothers.
Figure (3) Distribution of the study sample by their place of residence.

Shows that (78.6%) of mothers come from urban areas.
Figure (4) Distribution of study sample according to their family like plant food or animal diet rich vitamin A.

Figure (4) shows (64.3%) like plant food rich in vitamin A and (35.7%) like animal diet rich in vitamin A.
Table (4-1-3) Distribution study sample according to their dealing with children refuse food rich in vitamin A and knowledge regarding vitamin A deficiency.

(No.70)

<table>
<thead>
<tr>
<th>Item</th>
<th>correct</th>
<th>Uncorrected</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No</td>
<td>%</td>
<td>No</td>
</tr>
<tr>
<td>Deeling with child refuse food rich vitamin A</td>
<td>47</td>
<td>67.1</td>
<td>23</td>
</tr>
<tr>
<td>knowledge regarding vitamin A deficiency</td>
<td>62</td>
<td>88.6</td>
<td>8</td>
</tr>
</tbody>
</table>

Table(4-1-3): shows that (67.1%) of the sample responded with correct answers dealing with children refuse food rich in vitamin A and (88.6%) of them responded correctly regarding knowledge about vitamin A deficiency.
figure (5) Distribution of study sample according to their knowledge regarding sources of vitamin A.

figure (6) shows that (70%) from mothers' samples say fish and (20%) milk & meet (10%) are not know.
Figure (6) Distribution of study sample according to mothers knowledge regarding prevention of vitamin A deficiency.

Figure (7) shows that (77.1%) from mothers sample uncorrect answers regarding knowledge from prevention to vitamin A deficiency (22.9%) correct answers.
Table (4-1-4) Distribution of study sample according to their knowledge about sings and symptoms and complication of vitamin A deficiency.

<table>
<thead>
<tr>
<th>Item</th>
<th>Correct</th>
<th>Uncorrect</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No</td>
<td>%</td>
<td>No</td>
</tr>
<tr>
<td>Night blindness</td>
<td>62</td>
<td>88.6</td>
<td>8</td>
</tr>
<tr>
<td>Skin and hair dryness</td>
<td>53</td>
<td>75.7</td>
<td>17</td>
</tr>
<tr>
<td>Conjunctiva in the eye</td>
<td>65</td>
<td>92.9</td>
<td>5</td>
</tr>
<tr>
<td>Growth retardation</td>
<td>51</td>
<td>72.9</td>
<td>19</td>
</tr>
<tr>
<td>Dental decay</td>
<td>36</td>
<td>51.4</td>
<td>34</td>
</tr>
<tr>
<td>Death</td>
<td>42</td>
<td>60</td>
<td>28</td>
</tr>
</tbody>
</table>

Table (4-1-4) shows that (88.6%,75.7%,72.9%) from mothers samples correct answers regarding night blindness, skin and hair dryness and growth retardation is complication of vitamin A deficiency and (51.4%) of them responded correctly regarding Dental decay.
Table (4-1-5)  Distribution study sample according to their knowledge about benefits of vitamin A (No.70)

<table>
<thead>
<tr>
<th>Item</th>
<th>Correct</th>
<th></th>
<th>Uncorrect</th>
<th></th>
<th>Total</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No</td>
<td>%</td>
<td>No</td>
<td>%</td>
<td>No</td>
<td>%</td>
</tr>
<tr>
<td>Improve vision at night</td>
<td>47</td>
<td>67.1</td>
<td>23</td>
<td>32.9</td>
<td>70</td>
<td>100</td>
</tr>
<tr>
<td>Strong bone and teeth</td>
<td>61</td>
<td>87.1</td>
<td>9</td>
<td>12.9</td>
<td>70</td>
<td>100</td>
</tr>
<tr>
<td>Prevent skin cancer occur</td>
<td>45</td>
<td>64.3</td>
<td>25</td>
<td>35.7</td>
<td>70</td>
<td>100</td>
</tr>
<tr>
<td>Improve immunity</td>
<td>52</td>
<td>74.3</td>
<td>18</td>
<td>25.7</td>
<td>70</td>
<td>100</td>
</tr>
</tbody>
</table>

Table (4-1-5) shows that (67.1%) from mothers samples correct answers regarding benefits of vitamin A Improve vision at night and (35.7%) of them responded incorrectly regarding benefit of vitamin A prevent skin cancer.
Table (4-1-6) Distribution of study sample according to mothers knowledge about diseases cause vitamin A deficiency and effect vitamin A deficiency on the children. 

(No.70)

<table>
<thead>
<tr>
<th>Disease</th>
<th>correct</th>
<th>Uncorrect</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No</td>
<td>%</td>
<td>No</td>
</tr>
<tr>
<td>Chronic diarrhea</td>
<td>52</td>
<td>74.3</td>
<td>18</td>
</tr>
<tr>
<td>Measles and chest infection</td>
<td>33</td>
<td>47.1</td>
<td>37</td>
</tr>
<tr>
<td>Malnutrition and liver cirrhosis</td>
<td>44</td>
<td>62.9</td>
<td>26</td>
</tr>
<tr>
<td>On the bone osteoporosis</td>
<td>45</td>
<td>64.4</td>
<td>25</td>
</tr>
<tr>
<td>On the eye blindness</td>
<td>62</td>
<td>88.6</td>
<td>8</td>
</tr>
<tr>
<td>Growth retardation</td>
<td>51</td>
<td>72.9</td>
<td>19</td>
</tr>
<tr>
<td>Dry skin and hiar</td>
<td>53</td>
<td>75.7</td>
<td>17</td>
</tr>
<tr>
<td>Congenital abnormalities</td>
<td>41</td>
<td>58.7</td>
<td>29</td>
</tr>
<tr>
<td>Dental decay</td>
<td>36</td>
<td>51.4</td>
<td>34</td>
</tr>
</tbody>
</table>

Table (4 – 1 - 6) shows that (74.3%, 47.1%, 62.9%) from mothers samples correct answers regarding Chronic diarrhea, Measles and chest infection, Malnutrition and liver cirrhosis and (64.4%, 88.6%, 72.9%, 58.7%) of them responded correctly regarding effect of vitamin A deficiency on the children on (bones, eyes, growth and congenital abnormalities).
Figure (7) Distribution of study sample according to mothers that complain of vitamin A deficiency before.

Figure (7) shows that (81.4%) from mothers samples no complain of vitamin A deficiency and (18.6%) complain before.
4.2 Discussion

Vitamin A deficiency is a major public health nutrition problem in the developing world. Especially affects young children, among whom deficiency can cause xerophthalmia and lead to blindness, limit growth, increase the risk of death. Vitamin A deficiency can extend through school age and adolescent years into adulthood.

Results of the current study revealed that more than half of the study sample (51.4%) their ages between 21 - 30 years, and the majority of them (78.6%) were living in urban areas, with relatively low educational level were (40%) either illiterate or only finished their primary schools. (61.4%) of the study sample were housewives (35.7%) their dialy income).

The study revealed that (59.2%) of participant mothers received educational program about nutrition and vit A they got their information about healthy food mainly from radio (22.4%) and from TV (58.5%) . This is differ from the study of (Mitra et al, 2012) were 61% of respondent mothers were participated in educational sessions.

Studies show that nutritional knowledge affects the quality of food intake and also healthy choices of purchased food (O’Brien & Davies, 2007), Advancement of individual nutrition knowledge (De Vriendt et al, 2009). One study showed that health advice encouraged expectant mothers to improve their food intake (Anderson et al., 1993).

Results of the current study revealed that most of participants has no knowledge about essential component of integrated nutrition except for milk ,meat and fish (55.7% and 20%) respectively and (10%) her don’t know.

Also the study revealed that (67.1%) had a good knowledge about the importance of vitamin A(respectively) for child and pregnant mothers,. This is similar to the results of (Karim et al, 2009) were 70.5% of respondents has adequate knowledge about the importance of vitamin A for children (Pearce ,2007).

The study revealed that the majority of respondents had adequate knowledge about the problems associated with vitamin A deficiency. This is different from study of (Mitraetal, 2012) were more than 88% had a good knowledge about these problems (specially for night blind ness and growth abnormalities).

When discussion sources of vitamin A the study also revealed that knowledge of respondents mothers was poor regarding different natural sources of vitamin A. (Karin et al, 2009).
Chapter Five

Conclusion and Recommendation
5- Conclusion and Recommendations

5-1 Conclusion

Most of the mothers in this study are lacking the adequate knowledge and awareness about the consequences of inadequate nutrition and important of vitamin A. for children. growth compared to urban mothers. There is a significant association between mothers’s knowledge of nutrition to the children.
5-2 Recommendations:
- The study recommended that a teaching program for mothers and other health center providers improve the children and mothers nutritional knowledge and eventually improve the health status of the mothers and children especially in rural areas.
- Mothers should attend healthy clinic in order to know their nutritional status about children.
- Child health centers should be made accessible to our communities to help in educating and preventing malnutrition and vitamin A deficiency in children.
References:

6- Curhan, GC; Willett, WC; Rimm, EB; Stampfer, MJ (2003).".


Appendix

بسم الله الرحمن الرحيم

إسبيبان عن معرفة الأمهات عن نقص فيتامين (أ) عند الأطفال دون سن الخامسة في مستشفى ود
مدني التعليمي للأطفال (2015).

1. العمر:
   - أقل من 20 سنة ( )
   - 21-30 سنة ( )
   - أكثر من 30 سنة ( )

2. مكان السكن:
   - المدينة ( )
   - الريف ( )

3. المستوى التعليمي:
   - أمية ( )
   - أساس ( )
   - ثانوي ( )
   - جامعي ( )

4. الوظيفة:
   - ربة منزل ( )
   - موظفة ( )
   - طالبة ( )
   - أخرى حددي ...........

5. الدخل الشهري:
   - أقل من 500 جنيه ( )
   - 500-1000 جنيه ( )
   - 1000-1500 جنيه ( )

6. المسؤول عن مصاريف الأكل في الأسرة:
   - الأب ( )
   - الأم ( )

7. المسؤول عن اختيار الأغذية في الأسرة:
   - الأم ( )
   - الأب ( )

8. الأطعمة المشروعة لدى الأسرة:
   - الأطعمة النباتية ( )
   - الأطعمة الحيوانية ( )

9. الأطعمة غير المشروعة لدى الأسرة:
   - الأطعمة النباتية ( )
   - الأطعمة الحيوانية ( )

10. كيفية التصرف في حالة رفض تناول الأطعمة الغنية بالفيتامين (أ):
    - أ/ التوجه إلى أقرب مركز صحي: صحيح ( )
    - ب/ تناول أقراص فيتامين (أ): صحيح ( )

60
11. كيفية التصرف في حالة نقص فيتامين (أ)
أ/ تناول الأطعمة الغنية بفيتامين (أ) صحيح ( ) خاطئ ( )
ب/ تناول أقراص فيتامين (أ) صحيح ( ) خاطئ ( )

correct ( )

12. نقص فيتامين (أ) هو خلل في امتصاص الجسم لفيتامين (أ)
صحيح ( ) خاطئ ( )

13. هل تلقيت تثقيف صحي عن فيتامين (أ) من المصادر التالية:
الراديو ( ) التلفاز ( ) محاضرات تثقيفية ( )
صحيح ( ) خاطئ ( )

14. من مصادر فيتامين (أ) إلى: 
اللبن واللحوم ( ) الأسماك ( ) لا أعرف ( )
صحيح ( ) خاطئ ( )

15. يؤدي نقص فيتامين (أ) إلى:
العشى الليلي ( ) جفاف الجلد ( )
صحيح ( ) خاطئ ( )

16. هل يؤثر نقص فيتامين (أ) على العيون:
صحيح ( ) خاطئ ( )

17. هل يؤثر نقص فيتامين (أ) عند الأطفال في:
أ/ وقف النمو لدى الأطفال صحيح ( ) خاطئ ( )
ب/ الجلد والشعر الساطع صحيح ( ) خاطئ ( )
صحيح ( ) خاطئ ( )

18. هل الطفل المصاب يكون عرضه للاصابة بأمراض أخرى
صحيح ( ) خاطئ ( )

19. أعراض نقص فيتامين (أ):
تشقق الجلد ( ) عدم المقدرة على الرؤية ليلا ( ) لا أعرف ( )
صحيح ( ) خاطئ ( )

20. فوائد فيتامين (أ):
أ/ يحفز المناعة ويحسن الرؤية الليلية صحيح ( ) خاطئ ( )
ب/ يقوي العظام عند الأطفال ويساعد في نمو العضلات صحيح ( ) خاطئ ( )
صحيح ( ) خاطئ ( )

21. المصادر الحيوانية لفيتامين (أ):
الكبد، صفار البيض، اللبن، زيت السمك صحيح ( ) خاطئ ( )
لا أعرف ( )

22. المصادر النباتية لفيتامين (أ):
أ/ الخضروات الخضراء، القمح صحيح ( ) خاطئ ( )
لا أعرف ( )
ب/ هل تناولتي أقراص (حبوب) فيتامين (أ) أثناء الحمل: نعم ( ) لا ( )

23. من تأثير نقص فيتامين (أ) على الجنين:

يؤدي إلى التشوهات الخلقية ونقص وزن الجنين صحيح ( ) خطا ( )

24. من الأسباب التي تؤدي إلى نقص فيتامين (أ) عند الأطفال:

أ/ الأسهال المتكرر صحيح ( ) خطا ( )

ب/ الحصبة والتهاب الجهاز التنفسي صحيح ( ) خطا ( )

ج/ سوء التغذية وتليف الكبد صحيح ( ) خطا ( )

25. مضاعفات نقص فيتامين (أ):

الاصابة بالعمى، جفاف الجلد، تسوس الأسنان صحيح ( ) خطا ( )

26. الوقاية من نقص فيتامين (أ):

أ/ تناول الأغذية الغنية بفيتامين (أ) صحيح ( ) خطا ( )

ب/ تناول أقراص فيتامين (أ) صحيح ( ) خطا ( )

27. يؤدي نقص فيتامين (أ) إلى الوفاة:

صحيح ( ) خطا ( )

28. هل عانيتني من نقص فيتامين (أ) من قبل:

نعم ( ) لا ( )

( )