Ocular manifestations of severe protein energy malnutrition in Sudanese children

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To the memory of my father
who paved my way to
the profession I admire most.

and to

my mother who monitored
my passage throughout.
I would like to express my thanks and deep gratitude to my supervisor Dr. Abdul Rahman Diab, Faculty of Medicine, University of Khartoum, for his wise guidance, valuable advise, which contributed very much to the completion of this thesis.

It is a pleasure to acknowledge the help I have received from Dr. Amel Aoun Elshareif, Assistant Prof. of Ophthalmology, Co-supervisor for her patience, meticulous understanding and scholarly discussion.

My thanks and respect to Dr. Mohamed Nour Hassan for suggesting this work; and Dr. Elfatih Elfadl Bushara for his valuable suggestions and comments.

I owe a great deal to my mother in law Dr. Nimat Mohammed Elamir, my family and my husband Dr. Anis Elhadi Elzein, my sister Dr. Amna for their sacrifices and understanding, without which this work would not have been possible.

Thanks for all young co-operative patients and mothers.

ABBREVIATIONS

<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Meaning</th>
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<tr>
<td>K.C.E.H</td>
<td>Khartoum Children’s Emergency Hospital</td>
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<tr>
<td>Kwash</td>
<td>Kwashiorkor</td>
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<tr>
<td>M.U.A.C</td>
<td>Mid upper arm circumference</td>
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<tr>
<td>N.C.H.S</td>
<td>National Center for Health Statistics</td>
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<td>P.E.M</td>
<td>Protein energy malnutrition</td>
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Protein energy malnutrition is one of the leading causes of morbidity and mortality, worldwide, in childhood.

**Aims:** To study the prevalence of different eye findings in severely malnourished children, and to detect some of the risk factors associated with ocular manifestations in severe malnutrition.

**Method:** A total of 150 eyes of 75 patients with severe P.E.M who were admitted to Khartoum Children’s Emergency Hospital, were evaluated in a cross-sectional descriptive study for the ocular manifestations of P.E.M. Patient’s mothers or caretakers were interviewed for the present and past ocular and medical history. Ocular adnexa and anterior segment of the eye were examined in a good diffuse light using a pen torch and a
magnifying lens. After full mydriasis, the posterior segment was examined using a direct ophthalmoscope for every patient.

**Results:** The age of the patients ranged from 4-60 months and they were evenly distributed according to sex. 46.67% of the patients had lid oedema, 45.33% had lid ecchymosis; both findings were found statistically related to the nutritional status of the children, while other findings were not related. Conjunctivitis was found in 46.67% of patients and that might be due to decreased immunity among the study group. Trachoma was found in 14.67% and had no direct relation to the nutritional status of children. Signs of vitamin A deficiency were detected in only 4% of the study group. Retinal haemorrhages were found in 3 patients only, no other posterior segment findings were detected.

**Conclusions:** The ocular manifestations of severe protein energy malnutrition in Sudanese children are notable and comparable to findings reported in the literature.
المقدمة:

الأطفال في الوفاة المرضية أسباب اهمة من التغذية سؤญ مان.

الهدف:

في الأغداد التغذية سؤح مزاهر ودراسة تقصى بغرضا الدراسة هذه اجرية العناية بحال، الأطفال حواشان بمسطني العين.

الطريقة:

عدد العرضية المقطعية وصفية الدراسة هذه تتم 150 وسبيع لخمسة عين.

المريض تاريخهم عن المريض امته البسترية تم واقعي الفحص تم حيث للمرض ايكلينيكي فحص اجرى للعين.

الفحص اجرى العين منظور بمساعدة للعين الاصلية وعدة للمريض.

النتائج:

إلى اشهر اربعية من المريض اعمار تراوح 5 الذكور تساوي مع سنوات الموضوعة في واناث.

العيان اجفان في الوعور 46.67% وكسامت في الوعور 45.33%.

وجود بين التصادية الإحصائية علاقة ووجود والوقوع في الظاهرة الملتزمة وانتشار الالتهاب 46.67% المريض من التغذية بسوى الاصابيين لدى النقص في حالة.

الفيتامين نقصية علامات) ( لكل)

فقط مرضي ثالثة عند وجدت)

4.2% علامات تلاحظ على المريضة في التصلب في الفطر البصري أو الاكثرية في النزيف المانية الاكثرية (4)

الخلاصة:

بالملاحظة جديرة العين في التغذية سؤح مرض مزاهر تعتبر.
## LIST OF TABLES

| Table 1-1: Classification of xerophthalmia | Page 18 |
| Table 3-1: Distribution of patients with P.E.M according to age and sex | Page 39 |
| Table 3-2: Number and percent of ocular complaints among the study sample | Page 40 |
| Table 3-3: Number and percent of ocular findings among the study group | Page 41 |
| Table 3-4: Prevalence of trachoma among the study group | Page 42 |
| Table 3-5: Prevalence of vit. A deficiency signs among the study group | Page 43 |
| Table 3-6: Retinal findings among the study group | Page 44 |
| Table 3-7: Distribution of ocular finding in relation to nutritional status | Page 45 |
| Table 3-8: Distribution of ocular findings in relation to mother education | Page 46 |
| Table 3-9: Distribution of ocular findings in relation to vaccination | Page 47 |
| Table 3-10: Distribution of toilet type used by P.E.M children | Page 48 |
| Table 3-11: Distribution of water supply to patients of P.E.M children | Page 49 |
LIST OF FIGURES

Page

Fig. 1-1: Causes of corneal ulcers in malnourished children 9

Fig. 1-2: Causes of vit. A deficiency and vit. A metabolism 10

Fig. 1-3: Reactions of visual cycle in rod photoreceptors 11

Fig. 1-4: The fate of a single dose of vit. A 12

Fig. 3-I: Distribution of patients with PEM according to sex 50

Fig. 3-II: Distribution of patients with PEM according to their nutritional status 51

Fig. 3-III: Vaccination among the study sample 52

Fig. 3-IV: Relation between water supply and trachoma 54

Fig. 3-V: Relation between toilet type and trachoma 55
CONTENTS

Page

Dedication .........................................................................................I
Acknowledgement ...........................................................................II
Abbreviations ..................................................................................III
Abstract ...........................................................................................IV

Arabic abstract ................................................................................VI
List of tables .....................................................................................VII
List of figures ........................................................................................................... VIII

CHAPTER ONE

Introduction and literature review................................................................. 1
Justifications......................................................................................................... 2
6
Objectives........................................................................................................... 27

CHAPTER TWO

Patients and methods
........................................................................................................... 28

CHAPTER THREE

Results ................................................................................................................. 34

CHAPTER FOUR

Discussion......................................................................................................... 56
Conclusion ......................................................................................................... 61
Recommendations ............................................................................................. 62
References......................................................................................................... 63
Appendix (Questionnaire)
INTRODUCTION & LITERATURE REVIEW

1.1. General consideration

Malnutrition is one of the leading causes of morbidity and mortality, worldwide, in childhood. It can be due to in proper or inadequate food intake or may result from inadequate absorption of food. Insufficient food supply, poor dietary habits, food fadism and emotional factors may limit intake, certain metabolic abnormalities may also cause malnutrition.\(^{(1)}\)

Children as they are in the process of growth and development, less prepared to face the adverse forces of environment are particularly prone to suffer the unfavourable ecological conditions more than adult population. In particular case of nutritional deficiencies they have proportionately higher nutritional requirements than adults for most essential nutrients. All these factors explaining why nutritional deficiencies are, at the present time, one of the major health problems in children.\(^{(1,2)}\)

PEM occurs characteristically in children under 5 years, wherever the diet is poor in protein and energy. No age is immune, but in older persons the disease is much less frequent and the clinical manifestations not so obvious and usually less severe.\(^{(3)}\)

Deficiencies of retinol, folate, iron, magnesium and potassium are commonly found in P.E.M and may be the presenting clinical feature. Deficiency of retinol is the most important as it may lead to keratomalacia and permanent blindness. Research reports suggest that lack of zinc, copper, chromium, pyrodoxine, vitamins E and K and essential fatty acids may each be important in some circumstances. Thus in individual cases the cause of the child’s disease and the clinical features may vary greatly.\(^{(3)}\)

1.2. Definition:

Protein energy malnutrition is defined as a range of pathological conditions arising from a coincident lack, in varying proportion of proteins and calories, occurring most frequently in infants and young children.\(^{(1,2)}\)
Of the five broad classes of solid nutrients, recognized as basic to human nutrition (protein, carbohydrates, fat, minerals and vitamins), the first and last are most conspicuous; in clinical terms, when absent from the diet.\(^4\)

Mild to moderate, or first and second degree P.E.M is characterized primarily by growth retardation and reduction in motor activity. Severe P.E.M (i.e. marasmus, kwashiorkor, marasmus-kwashiorkor) presents striking biochemical changes and clinical signs.

1.3. Protein Energy Malnutrition global experience:

It is now recognized that some 56% of children deaths in developing countries are attributable to malnutrition potentiating effects.\(^5,6\)

In 1995, it has been estimated that currently more than 200 million children under five years are malnourished. This means that more than 30% of the world’s children aged under five years are still malnourished in terms of being under weight.

The overall mortality of severe malnourished patients is \(25 - 28\)% which is usually related to dehydration and severe infections.\(^7\)

In Cameron, nutritional status was estimated by categories based on weight for age and 6% were found to be severely malnourished.\(^8\) One study in Central Brazil showed that in children less than 5 years, 34% were malnourished, only 2% were severely malnourished.\(^9\) A nutritional status in survey of children age 0-5 years was carried out in Malawi, the overall prevalence of protein energy malnutrition was \(14\)%.\(^10\)

1.4. The Magnitude of P.E.M. in Sudan:

Efforts to know different aspects of malnutrition in Sudan has been started in the early sixties.\(^11\)

In a survey in Haj Yousif, Bushara found that the percentage of marasmus was 3.9% and Kwashiorkor was 2.6% of the total children.\(^12\) Zumrawi, et al., studied infant longitudinally from birth to one year in urban poor neighbourhoods in Khartoum Province, 50% of children were found to be undernourished.\(^13\)
In a survey conducted by the Ministry of Health between 1986 – 87, it was found that 32.1% of children under 5 years were stunted, 14.1% were under weight and 1.7% having severe malnutrition.\textsuperscript{(14)} Other surveys of Ministry of Health, Sudan showed that under nutrition was a prevalent problem among most of the provinces ranging from 10.2% in Khartoum to 26.6% in Sennar State.\textsuperscript{(15)}

In 1997 in Khartoum State, a nutritional survey among 2775 children below 5 years revealed that 16.6% were malnourished, 3.4% of these children were severely malnourished.\textsuperscript{(16)}

1.5. Classification of childhood malnutrition:

The widely used classification of childhood malnutrition are:

1- \textbf{Gomez classification}:

Used widely in which the weight for age of a patient is calculated according to standard as follows:

i- Normal patients having more than 90% of the expected weight for age.

ii- Mild (first degree malnutrition) having 89% -75% of the expected weight for age.

iii- Moderate (second degree malnutrition) having 74-60% of the expected weight for age.

iv- Severe (third degree) in which patients either have less than 60% of the expected weight for age or have lower limbs oedema.\textsuperscript{(17)}

2- \textbf{Welcome Trust Classification}:

It uses weight for age and the presence of nutritional oedema to classify the nutritional status of children.
1. Weight for age less than 60% without oedema is marasmus, with oedema is marasmic-kwashiorkor.

2. Weight for age 61-80% without oedema is underweight, with oedema is kwashiorkor.

3. Weight for age more than 80% without oedema is well nourished child and with oedema is kwashiorkor.\(^{(17)}\)

3- **Waterlow classification:**

It classifies stunting, which represents chronic malnutrition, into three degrees using height for age:

i- More than 95% is normal.

ii- First degree 95-90%.

iii- Second degree 89-85%.

iv- Third degree less than 85%.

Also it called the present malnutrition, wasting, measured by loss of weight related to height. If the weight for height:

i- More than 90% there is no wasting.

ii- 90-80% there is first degree wasting.

iii- 80-70% there is second degree wasting.

iv- Less than 70% there is third degree wasting.\(^{(17)}\)

4- **Use of mid upper arm circumference to classify malnutrition (M.U.A.C) :**

Mid upper arm circumference had been proposed as an alternative index of nutritional status in emergency stations such as famines or refugee crises. In community based studies, M.U.A.C appears superior predicator of childhood mortality compared with height and weight.

i- Reading > 13.5 cm, adequately nourished children.

ii- Between 13.5 – 12.5, moderately malnourished children.

iii- Below 12.5 cm indicates severe malnutrition.

1.6. **Symptoms and signs of severe malnutrition:**
According to welcome classifications these are:

**a- Marasmus:**

(Greek marasmus, wasting). Patients presents with failure to gain weight, irritability or apathy. The marasmic child is often less than one year, there is no or little subcutaneous fat, loose skin, and muscles are markedly wasted.

**b- Kwashiorkor:**

Means “deposed child” that is the child no longer suckled, it may become evident from early infancy to about 5 years of age, usually after weaning from the breast. It the presenting symptoms usually include oedema, unhappiness or apathy anaroxia, vomiting, skin and mucous membrane changes. Skin changes consist of hyper and hypopigmented areas, desquamation and ulceration. A further more serious manifestation is that of weeping dermatitis resembling burns. Chilosis and angular stomatitis are common. The extremities are frequently cold and the liver is enlarged with fatty infiltration.(17)

Secondary immunodeficiency is one of the most serious and constant features.

**c- Marasmic Kwashiorkor:**

This is a syndrome, which has the characteristics of both Kwashiorkor and marasmus. These children present with weight for age is less than 60%. In addition they have lower limbs oedema.(18)

**1.7. Protein Energy Malnutrition and the Eye:**

The ocular manifestations of P.E.M are non-specific and manifest as lid oedema, chemosis, and predisposition to external infection. Keratopathies of varying severity have also been described, but in many patients these may be due to associated vitamins deficiency.(4)

**1.2.7. Vitamin A:**

The chemical name of vitamin A is retinal and it is a fat soluble unsaturated alcohol, which is an essential vitamin.
The word xerophthalmia-dry eye-means precisely the changes in the eye from vitamin A deficiency. Many children with vitamin A deficiency are malnourished in other ways, and in particularly common and important cause of corneal ulcers, especially in Africa. There are also some other causes of these ulcers, which affect only the eye, and not the rest of the body. These are:

i- Local infections to the cornea. This may be from a virus (in particular the measles virus itself or herpes simplex) or from a bacteria.

ii- Exposure damage to the cornea from poor eyelid closure.

iii- Toxic traditional medicines applied to the eye.\(^{19}\)

**Fig.1.1:** Corneal ulcers in malnourished children are usually caused by several different factors working to damage the cornea. Some of the more important are shown in the diagram.
**Fig. 1.2:** shows the left side of the diagram shows how vitamin A passes from food to the eye in a healthy person. The right side shows the mean causes of vitamin A deficiency these are indicated by circles.\(^{(19)}\)
Fig. 1.3: ..............
Dietary sources of retinal:

There are two sources: animal foods and plant foods. Animal foods contain the active vitamin retinal, but some foods are much richer in retinal than others. The liver, milk products are also very rich, it can also be found in kidney, eggs and meat.

Plant foods contain carotene pigments, which is chemically related to retinal. The best source of vitamin A is red palm oil, green leafy vegetables (e.g. spinach) and orange-coloured fruit and vegetables (e.g. carrots, mangoes).\(^{(19)}\)

Fig. 1.4: *The fate of a single dose of vitamin A:*

![Diagram of the fate of vitamin A](image)

Dietary vit. A  Not absorbed 20%

Absorbed 80%  Conjugated and excreted in faeces 20-30%

Oxidized and excreted in urine (10-20%)

Stored in the liver 30-50%.

Functions of vitamin A:
The maintenance of healthy epithelial tissues is the most important function of vitamin A. There are two specific changes noted in epithelial tissues in vitamin A deficiency:

i- Keratinization of the epithelium, keratin is a hard protein present in the skin. In xerophthalmia it develops on the surface of the mucous membranes as well. These then become harder and resist wetting.

ii- Loss of goblet cells. These cells secrete mucous which helps maintain the moistness and wettable of mucous membranes, which is reduced in xerophthalmia.

iii- The formation of visual purple is only the function of vitamin A, which had been defined biochemically. The aldehyde form of vitamin A is called “retinal”. Retinal combines biochemically with opsin (aprotein found in the photosensitive part of the rods in the retina) to form visual purple, when the retinal molecule is expressed to light, it changes from one chemical form (isomer) to another. Further chemical changes then produce the electrical impulse, which the brain eventually interprets as light. Vitamin A deficiency produces a condition called “night blindness”. In night blindness dark adaptation and the ability to see in the dark are specifically diminished.\(^{19}\)

**Vitamin A deficiency in young children:**

Young growing children are more at risk from vitamin deficiency than adult because:

i- The vitamin A requirements per unit of body weight is much greater than an adult. The recommended daily intake of vitamin A per kg body weight is 65 µg (125 I.U) for an infant, but only 12 µg (36 I.U) for an adult.

ii- A child can not store vitamin A in the liver as well as an adult.\(^{19}\)

**Signs and symptoms of vitamin A deficiency in the eye:**

The primary signs are specific for xerophthalmia. They start with the least severe and end with the most severe.

The secondary signs are non-specific for xerophthalmia, but are likely to indicate vitamin A deficiency.
XIA conjunctival xerosis:

Changes occur on the bulbar conjunctiva, especially in the exposed parts, these are:

- Dryness causes the conjunctiva to lose its shiny luster and look like a wax or paint, it also becomes “unwettable”, so that the tear film breaks up and leaves dry patches. As the conjunctiva thickens and loses its transparency, the underlying blood vessels are more difficult to see.

- Increased pigmentation gives the conjunctiva a fine, diffuse, smoky, grey-brown appearance, especially near limbus in the inter palpebral fissure. This pigmentation also occurs in vernal conjunctivitis.

- A creamy white debris is sometimes found, especially where the upper and lower eyelids meet, the lower fornix, or the lid margin around the openings of the meibomian glands. This debris is a feature of advanced conjunctival xerosis.

XIB Bitot’s spots with conjunctival xerosis:

A Bitot’s spot is a small plaque of material on the surface of the bulbar conjunctiva, and nearly always in the inter palpebral fissure. The material is usually foamy, but may look waxy or greasy, and may contain pigment from eye make-up. If the material is wiped away, it leaves a dry conjunctival bed with a rough surface. A Bitot’s spot usually occurs temporal to the cornea, but it is occasionally nasal. It is either oval in shape or forms a triangle pointing away from the cornea.

X2 corneal xerosis:

Corneal xerosis is an extension of the conjunctival signs on to the cornea. It is much less common than conjunctival xerosis, but it is a highly specific sign. It has characteristic superficial punctate lesions of the inferior nasal aspect of the cornea that stain brightly with fluorescein. More severe lesions become more numerous and spread upwards over the central cornea, and the corneal stroma becomes oedematous.

Clinically the cornea develop classical xerosis, a hazy, blisterless, dry appearance, first appeared near the limbus. Thick, keratinized plaques resembling Bitot’s spot may form on the corneal surface, these are often densest in the interpalpebral zone.
One of the remarkable features of xerophthalmia is that the changes up to this stage are rapidly and completely reversible with vitamin A treatment. After only 3-4 days conjunctival and corneal xerosis and night blindness will be completely cured.

**X3A corneal ulceration with xerosis:**

The following features are characteristic of corneal ulceration from xerophthalmia:

I- Both conjunctival and corneal xerosis are present.

II- Both eyes are diseased to some extent.

III- The ulcers are in the central and lower part of the cornea and typically lacks the grey, infiltrated appearance of ulcers of bacteria origin.

Often there is very little pain, tissue reaction or inflammation. However, if there are any signs at all of corneal ulceration, urgent treatment is necessary.

**X3B keratomalacia:**

Localized keratomalacia is a rapidly progressive condition affecting the full thickness of the cornea. It first appear as an opaque grey to yellow mound or out pouching of corneal surface. In more advanced disease the necrotic stroma sloughs leaving a large ulcer or descematocele. As with smaller ulcers they are usually peripheral and heals as dense, white, adherent leukomas.

**XN night blindness:**

Is the most sensitive sign of vitamin A deficiency, and usually appears before any conjunctival or corneal changes.

**XF xerophthalmia fundus:**

Vitamin A deficiency seems to produce a characteristic change in the retina. Pale yellow spots appear, especially near the course of the retinal vessels and also in the retinal periphery.

**XR xerophthalmic scars:**
Healed sequel of prior corneal disease related to vitamin A deficiency include opacities or scars of varying density (nebula, macula, leukoma) weakening and out pouching of the remaining corneal layers (Staphyloma, descematocele) and were loss of intraocular contents had occurred, phthisis bulbi, a scared shrunken globe.\textsuperscript{(19,20)}

Table No. 1: Classification of xerophthalmia\textsuperscript{(19)}

<table>
<thead>
<tr>
<th>Primary signs:</th>
<th>Secondary signs:</th>
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<tr>
<td>X1 A</td>
<td>Conjunctival xerosis</td>
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<tr>
<td>X1 B</td>
<td>Bitot’s spots with conjunctival xerosis</td>
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<tr>
<td>X2</td>
<td>Corneal xerosis</td>
</tr>
<tr>
<td>X3 A</td>
<td>Corneal ulceration with xerosis</td>
</tr>
<tr>
<td>X3 B</td>
<td>Keratomalacia</td>
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1.9. Vitamin B deficiency:
Vitamin B is a collective term for a dozen different dietary factors that are grouped together because of certain common features such as water solubility and high concentration in liver and yeast.

**Thiamin deficiency:**

Manifest by progressive, peripheral neuropathy involving sensory and motor function, cardiac decompensation and personality changes, the complete syndrome is known as (Beriberi). Ocular abnormalities associated with beriberi include central scotomas and external ophthalmoplegias affecting particularly cranial nerves III and VI. \(^{(4)}\)

**Riboflavin deficiency:**

Is of special concern to the ophthalmologists because of corneal neovascularization that is one of the earliest signs. Initially, there is congestion of the limbal plexes, followed by invasion of the stroma through the entire corneal circumference. The patient notes, burning sensation, lacrimation and photophobia. Oral administration of riboflavin results in prompt resolution of the keratitis, although ghost vessels persist through out life. Other ocular conditions in which riboflavin deficiency has been implicated include phlyctenular keratoconjunctivitis and rosacea keratitis. Similarly the pannus of trachoma is recognized as being exacerbated by malnutrition, and it may be ameliorated by riboflavin therapy.\(^{(4)}\)

**Nicotinic acid deficiency:**

Pellagra is the disease resulting from the dietary deprivation of niacin and its amino acid tryptophan. The disease is characterized by dermatitis and various neurological and gastrointestinal disorders. The dermatitis initially resembles a first degree burn and progress to hyperpigmentation and hyperkeratosis particularly in the exposed areas including the face and eyelids. In the optic neuritis and pigmentary maculopathy with variable losses of visual acuity and field have been reported.\(^{(4)}\)

**Vitamin B12:**

Cyanocobalamin and folic acid are necessary as coenzymes in the initial phases of D.N.A synthesis deficiency leads to megaloblastic anaemia, if the anaemia is severe enough, the ocular fundus will reveal retinal haemorrhages, exudates and congested vessels quite
similar to those anaemia of any aetiology. The unique aspect of vitamin B12 deficiency is the retrobulbar neuritis, which is part of the associated syndrome of subacute combined degeneration (in which there is change to the myelinated fibres of the posterior and lateral horns of the spinal cord resulting in parathesia, ataxia and spastic paraplegia); resulting initially in central scotoma, which if not treated may progress to optic atrophy.\textsuperscript{4}

1.10. Vitamin C deficiency:

Vitamin C is essential in intracellular oxidation reduction reaction, particularly in mesenchymal cells which synthesis collagen. The deficiency leads to scurvy, the ocular manifestations of which consist of subconjunctival haemorrhages, although in advanced case hyphaemas, retinal haemorrhages have been found.\textsuperscript{4}

1.11. Vitamin D deficiency:

Vitamin D is fat-soluble vitamin, not only produced by the u/v irradiated dermis but also by activated macrophages. The vitamin is a potent inhibitor of T cell proliferative and of the cytokines production. This generates a neat feedback loop at sites of inflammation where macrophages activated by IFN-\(\alpha\) produce vitamin D which suppress the T-cells making the interferon.\textsuperscript{4}

1.12. Immunity in Malnutrition:

Immunodeficiency disease results from the absence or failure of normal function, of one or more elements of the immune system specific immunodeficiency diseases involve abnormalities of T or B cells, the cells of the adaptive immune system. Non-specific immunodeficiency diseases involve abnormalities of elements such as complement or phagocytes, which act non-specifically in immunity. Primary immunodeficiency disease are due to intrinsic defects in cells of the immune system and are for the most part genetically determined secondary immunodeficiency diseases result from extrinsic factors, such as drugs, irradiation, malnutrition or infection.\textsuperscript{21}

Worldwide, protein energy malnutrition is probably the leading cause of immunodeficiency in children.
The immune responses of the body are produced by cells arising in the thymus, lymph nodes and spleen, the lymphoreticular organs. These are very immature at birth and develop rapidly in the first two years of life.\(^{(19)}\)

In both marasmus and kwashiorkor the thymus, tonsils, spleen and other lymphoid tissues are atrophied. These changes are accompanied by delayed or absent tuberculin response and other skin hypersensitivity reactions; reduced complement activity in the serum, especially the C3 component; reduced numbers of thymus dependent lymphocytes (T-cells) in the blood.

The depression of all mentioned is attributable mainly to protein deficiency but lack of zinc, folate and other nutrients are sometimes in part the cause that it is responsible for the high mortality from measles, gastroentritis and other common infection in malnourished young children seem beyond doubt.\(^{(22)}\)

Total circulating lymphocytes are reduced in number and also the T lymphocytes present have a poor response to in vitro mitogenic stimulator. Vitamin A deficiency produces reduced CD4/CD8 ratio and total CD4 naive T cells. This immunocompromised status improves with dietary rehabilitation, functional recovery occurs within 2 weeks and the total lymphocyte count recover within 4 weeks.\(^{(30)}\)

The concentration of B lymphocytes in lymphoid tissues and peripheral blood is normal immunoglobulin\(^{(19)}\) synthesis is increased as reflected in elevated total circulating IgA, IgM, IgG, IgD and IgE levels. The concentrations are greater in children with kwashiorkor than those with marasums.

Malnourished children have reduced secretary IgA (sIgA) levels in nasal washings, duodenal fluids, and tears despite the elevated level of IgA in the serum. The loss of secretory immunity may contribute to an increase in the respiratory, intestinal infections.

The response of polymorphonuclear (PMN) leukocytes to chemotactic stimulation is normal, but that of the macrophages is reduced in PEM.

All P.E.M subjects have depressed concentrations of specific complement factors (Ciq, Cis, CZ, Cs, C6, C8, C9 and C3 PA).\(^{(21)}\)
1.13.a. Protein energy malnutrition and anaemia:

Diet that lead to P.E.M is frequently lacking in iron, folic acid and other vitamin.\(^{(1,2,30)}\)

Changes of anaemia in the eye occurs in:

a- Conjunctiva which appears as pallor.
b- Retina.

1.13.b. Retinopathy in anaemia:

Retinal changes in anaemias are usually innocuous and rarely of diagnostic importance.

1- **Retinopathy:** is characterized by haemorrhages, cotton wool spots and venous tortuosity. Roth spots may also be seen in same cases.

a- The duration and types of anaemia do not influence the occurrence of these changes, which are more common when anaemia coexists with thrombocytopenia.
b- Flame-shaped haemorrhages and cotton-wool spots may occur in the absence of other haematological abnormalities.
c- Retinal venous tortuosity seem to be related to the severity of the anaemia and the reduction in haematocrit.
d- Both spots are intraretinal haemorrhages with white centers, which are thought to represent fibrin thrombus occluding a ruptured blood vessel. Both spots may also occur in bacterial endocarditis and leukaemia.

2- **Optic neuropathy:** With centrooccal scotoma in patients with pernicious anemia unless treated, permanent optic atrophy may insure pernicious anaemia also cause dementia, peripheral neuropathy and subacute combined degeneration of the spinal cord characterized by posterior and lateral columnn diseases.\(^{(23)}\)
JUSTIFICATIONS

1- Malnutrition is a common paediatric problem in Sudan with high morbidity and mortality.

2- The area of eye findings associated with PEM is deficient in research.
OBJECTIVES

The aims of this study is to:

3- Determine the prevalence of different ocular findings in severely malnourished Sudanese children age group 4-60 months.

4- To determine some of the risk factors associated with ocular manifestations in severe PEM, e.g.
   - Vaccination.
   - Indicators of home environment.
   - Education of the mother.
MATERIAL & METHODS

2.1 Setting:

This study was carried out in Khartoum Children’s Emergency Hospital (K.C.E.H). It is a central hospital that receives patients from different parts of Sudan and where a special ward of malnutrition is found.

Some patients were admitted in K.C.E.H and then referred by physicians to Khartoum Teaching Hospital (K.T.H).

Interviews and examinations were done in:

1- Khartoum Children’s Emergency Hospital, malnutrition ward.
2- Khartoum Teaching Hospital, Department of Paediatrics, malnutrition wards in C1, C2.

2.2. Study population:

All patients admitted with severe malnutrition ages 4-60 months who presented to the hospital on all days of the week including Fridays were interviewed and examined. In this age group (4-60 months) following weaning, protein energy malnutrition presents clinically.

Patients were diagnosed to be severely malnourished by paediatricians if they have any of these criteria:

1- Weight for age < 60% of NCHS.
2- Weight for height <70% of NCHS.
3- Presence of nutritional oedema.

These patients were classified into Mmaramus, Marasmic-Kwashiorkor and Kwashiorkor according to the welcome classification.

Inclusion and Exclusion criteria:
a) Patients included in the study: -

All patients age group 4-60 months having severe malnutrition and admitted to K.C.E.H.

b) Patients Excluded from the study: -

Any patient admitted for more than 48 hours was excluded from the study.

Some of the symptoms and signs of vitamin-A deficiency e.g. night blindness resolves within 24 – 48 hours, active conjunctival xerosis and Bittots spots begin to resolve within 2-5 days. Therapeutic dose of vitamin-A is given to any patient admitted to the malnutrition ward, so that any patient admitted for more than 48 hours was excluded from the study.

2.3. Procedures:

* Study design:

This is a descriptive, hospital-based study.

* Study tools:

The following tools were used to examine patients in malnutrition ward:

- Pen torche.
- Loupe.
- Pocket-type Keeler direct ophthalmoscope.
- Eye Drops: Tropicamide 1%, Tetracain E.D.
- Cotton balls.
- Fluorescein papers.
- E. type visual acuity chart.

Consent:

Verbal consent was taken from patients or accompanying caretakers of children in the study. Also verbal consents were taken from the treating paediatrician caring for these children.
Sample size: The sample size was determined according to the advice of a statistician. It was calculated according to the formula:

\[ n = \frac{Z^2 pq}{E^2} \]

- \( n \) = Sample size.
- \( Z \) = 1.96 (95% confidence limit).
- \( p \) = Prevalence.
- \( q = 1-p \)
- \( E \) = Correction factor.

Using this formula, with a prevalence of severe malnourish in Khartoum State, which was 3.4%, the minimum total sample size was found to be 50 patients (75 patients were examined in this study).

Methodology:

A questionnaire was designed (appendix); the questions were completed by asking the mother or caretaker. The questionnaire consisted of:

a) History:

This includes personal data as well as the present ocular and medical history.

b) Clinical examination:

1- Using E type visual acuity chart and the pinhole test, the visual acuity was obtained. The test was performed at six meters distance in the malnutrition ward. The visual acuity chart was illuminated by the room light. Any patient with visual acuity less than 6/6 was referred for the Refraction Department in Khartoum Teaching Eye Hospital for proper assessment.

Preverbal children were examined for V.A by:

i- Occlusion of one eye.

ii- Rotation test.

2- Ocular adnexa and the anterior segment of the eye, including conjunctiva, sclera, cornea, anterior chamber, pupil and lens were examined in a good diffuse light using a pen torch and a loupe. Particular attention was offered for signs of protein-energy malnutrition (lid oedema, chymosis, external infections) and vitamin-A deficiency.
3- The cornea were examined using the loupe; and stained with fluorescein after application of topical anaesthesia, if the normal luster of the cornea was lost.

4- Full mydriasis was obtained for every patient using 1% Tropicamide eye drops and the posterior segment was examined (with the help of the mothers) using a pocket type Keeler direct ophthalmoscope. Particular attention was offered for optic atrophy, optic neuritis, xerophthalmia fundus, macular pigmentation and retinal haemorrhages.

2.4. Data analysis:

The questionnaire was coded and a master sheet was constructed to arrange the raw data. IBM compatible computer was utilized for data analysis using SPSS (Statistical Package Social Sciences) programme. Chi-square test was used for categorized variables at 95% considered level (P= 0.05). Only P. value ≤ 0.05 was considered to indicate significance.

Computer program (Excel) was used for graphical presentations.

2.5 Limitation of the study:

- Although PEM is one of the leading causes of morbidity and mortality, worldwide, in childhood, Literature concerned with eye manifestations of malnutrition is few and research is scanty.

- Ophthalmic examinations is severely ill children with severe malnutrition was an inconvenient for mothers and care takers.
• Attendance of patients to Khartoum Teaching Eye Hospital, Out patient Department for further assessment was not always accomplished.

• Visual acuity could not be done for 23 patients, because they were very ill and uncooperative. Other tools for examination of visual acuity in preverbal children were not available.

RESULTS

A total of 150 eyes of 75 patients with severe PEM were examined in this study with particular attention exerted on the ocular manifestations of the disease and vitamins deficiency.

3.1. Demographic data:

Almost half of the study group was in the age group 4-18 months, this constitutes 37 patients (49.33%). Minor gender differences were found regarding differences in age groups. This is shown in Table 3.1.
The male to female ratio was found to be evenly distributed, 36 patients were males (48%) and 34 were females (52%). This is shown in Fig. 3.I.

3.2. Nutritional status:

Of the 75 patients with severe PEM included in the study 29 patients (38.66%) were marasmic kwash, 17 patients (36%) were marasmic, while 19 patients (25.34%) were Kwashiorkor (Fig. 3.II).

3.3. Vaccination among the study sample:

Thirty patients (40%) were fully vaccinated, 38 patients (50.67%) were partially vaccinated. Only 7 patients (9.39%) were not vaccinated at all. This is shown in Fig. 3.III.

3.4. The presenting eye complaints of children in the study:

The commonest presenting complaint was discharge 21 patients (28%). This was followed by itching 19 patients (25.33%). Photophobia in 6 patients (8%) and 9 patients (12%) complained of tearing. The remaining 20 patients (26.67%) had no complaints (Table 3-2).

3.5. Visual acuity:

Visual acuity was normal in 104 eyes of 52 patients examined. Verbal children examined, their visual acuity was 6/9 or better.

3.6. The number and percent of ocular findings among the study group:

Table 3-3 shows that lid oedema was found in 35 patients (46.67%), lid chemosis was found in 34 patients (45.33%), conjunctivitis was found in 35 patients (46.67), 11 patients (14.67%) of the study group had trachoma. Retinal haemorrhage was detected in 3 patients only (4%), 2 patients (2.67%) had not full ocular movement. Pallor was observed in 72 patients (96%).
3.7. Prevalence of vitamin-A deficiency among the study group:

Signs of vitamin-A deficiency was detected in 3 patients (4%), 2 patients with conjunctival xerosis (1.33%) and 2 patients (2.67%) had corneal xerosis (Table 3-5).

3.8. Retinal findings among the study group:

Table 3-6 shows that retinal haemorrhage was the only finding in the posterior segment and was found in 3 patients only (4%). No optic atrophy, xerophthalmia fundus, macular pigmentation, optic neuritis, Roth spots, venous tortuosity were detected.

3.9. Distribution of ocular findings in relation to nutritional status:

In Table 3-7, 17 patients (22.67%) with lid chemosis had kwashiorkor, another 17 patients had marasmic-kwash, no marasmic patient had lid chemosis (P = 0.001). Lid oedema was found in one patient with marasmus (1.33%), 17 patients (22.67%) marasmic kwash, 17 patients with kwashiorkor (P = 0.001) conjunctivitis was detected in 14 patients (18.67%) with marasmus, 9 patients (12.0%) with kwash, and 12 patients (16.0%) with maerasmic kwash (P = 0.07). Only lid oedema and lid chemosis had a significant relation to the nutritional status of the children.

3.10. Risk factors associated with ocular diseases:

3.10a. Distribution of ocular findings in relation to mother education:

Table 3-8 shows that, 2 patients (2.67%) with no full ocular movement had illiterate mothers (P= 0.85), 26 patients (34.67%) with lid chemosis their mothers were illiterate, 3 mothers completed Khalwa, 10 mothers (13.3%) were at the primary school, 2 mothers at the secondary school (P = 0.48). Dacrocystitis was found in one patient (1.33%) whose mother was university graduate (P= 0.4). Education of the mother was not found significantly related to other ocular findings among the study group.
3.10b. Distribution of ocular findings in relation to vaccination:

Table 3-9 shows that, lid chemosis was found in 13 patients (17.33%) fully vaccinated, 17 patients (22.67%), partially vaccinated, 4 patients not vaccinated (P= 0.79). Dacrocystitis found in one patient (1.33%) who was not vaccinated (P= 0.007). Dacrocystitis was the only ocular finding which was statistically significant related to vaccination.

3.11. Distribution of toilet type:

Table 3-10 shows that, 48 patients (64%) of the study group use a bore hole inside the house as a toilet, 18(24%) use a hole outside the house, only 7 patients (9%) had a flush toilet inside the house.

3.12. Distribution of water supply to patients of PEM:

Table 3-11 shows that, 45 patients (60%) buy water in barrel for their daily use, 14 patients (19%) had tap in the house.

3.13. The relation between the water supply and prevalence of trachoma:

Fig. 3. IV shows that, out of the 11 patients with trachoma (14.67%), 8(10.67%) buy water for their daily consumption, 2 patients (2.67%) got their water from a public tab and one patient (1.33%) got water from the river (P = 0.32).

3.14. The relation between the type of toilet and prevalence of trachoma:

Fig. 3.V shows that, 7 patients (9.33%) of the study group with trachoma use a pit latrine inside the house while the remaining 4 patients (5.33%) use a hole outside the house as a toilet (P = 0.500).
Table 3-1: Distribution of patients with P.E.M according to age and sex

<table>
<thead>
<tr>
<th>Age (in months)</th>
<th>Male</th>
<th></th>
<th></th>
<th>Female</th>
<th></th>
<th></th>
<th>Total</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No.</td>
<td>%</td>
<td>No.</td>
<td>%</td>
<td>No.</td>
<td>%</td>
<td>No.</td>
<td>%</td>
<td></td>
</tr>
<tr>
<td>4 – 18</td>
<td>17</td>
<td>22.7</td>
<td>20</td>
<td>26.67</td>
<td>37</td>
<td>49.33</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>19 – 32</td>
<td>13</td>
<td>17.3</td>
<td>17</td>
<td>22.67</td>
<td>30</td>
<td>40</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>33 – 46</td>
<td>3</td>
<td>4.00</td>
<td>1</td>
<td>1.33</td>
<td>4</td>
<td>5.33</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>47 – 60</td>
<td>3</td>
<td>4.00</td>
<td>1</td>
<td>1.33</td>
<td>4</td>
<td>5.33</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>36</td>
<td>48</td>
<td>39</td>
<td>52</td>
<td>75</td>
<td>100</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Table 3-2: Number and percent of the ocular complaints among the study sample

<table>
<thead>
<tr>
<th>Presenting complaints</th>
<th>No.</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Discharge</td>
<td>21</td>
<td>28.00</td>
</tr>
<tr>
<td>Itching</td>
<td>19</td>
<td>25.33</td>
</tr>
<tr>
<td>Photophobia</td>
<td>6</td>
<td>8.00</td>
</tr>
<tr>
<td>Tearing</td>
<td>9</td>
<td>12.00</td>
</tr>
<tr>
<td>No complaints</td>
<td>20</td>
<td>26.67</td>
</tr>
</tbody>
</table>
Table 3-3: Number and percent of ocular findings among the study group

<table>
<thead>
<tr>
<th>Ocular finding</th>
<th>No.</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>No full ocular movement</td>
<td>2</td>
<td>2.67%</td>
</tr>
<tr>
<td>Lid chemosis</td>
<td>34</td>
<td>45.33%</td>
</tr>
<tr>
<td>Lid oedema</td>
<td>35</td>
<td>46.67%</td>
</tr>
<tr>
<td>Pallor</td>
<td>72</td>
<td>96%</td>
</tr>
<tr>
<td>Conjunctivitis</td>
<td>35</td>
<td>46.67%</td>
</tr>
<tr>
<td>Dacrocystitis</td>
<td>1</td>
<td>1.33%</td>
</tr>
<tr>
<td>Trachoma</td>
<td>11</td>
<td>14.67%</td>
</tr>
<tr>
<td>Subconjunctival haemorrhage</td>
<td>1</td>
<td>1.33%</td>
</tr>
<tr>
<td>Corneal opacity</td>
<td>1</td>
<td>1.33%</td>
</tr>
<tr>
<td>Keratitis</td>
<td>2</td>
<td>2.67%</td>
</tr>
<tr>
<td>Retinal haemorrhage</td>
<td>3</td>
<td>4%</td>
</tr>
</tbody>
</table>

Table 3-4: Prevalence of trachoma among the study group

<table>
<thead>
<tr>
<th>Trachoma signs</th>
<th>No.</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>T.F</td>
<td>7</td>
<td>9.33%</td>
</tr>
</tbody>
</table>
Table 3-5: Prevalence of vitamin-A deficiency signs among the study group

<table>
<thead>
<tr>
<th>Vit. A deficiency signs</th>
<th>No.</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Conjunctival xerosis</td>
<td>1</td>
<td>1.33</td>
</tr>
<tr>
<td>Corneal xerosis</td>
<td>2</td>
<td>2.67</td>
</tr>
<tr>
<td>Other vitamin A deficiency signs</td>
<td>0</td>
<td>0.0</td>
</tr>
<tr>
<td>-------------------------------</td>
<td>---</td>
<td>-----</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td>3</td>
<td>4%</td>
</tr>
</tbody>
</table>

Table 3-6: Retinal findings among the study group

<table>
<thead>
<tr>
<th>Retinal</th>
<th>No.</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Retinal haemorrhage</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>Optic atrophy</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Xerophthalmia fundus</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Macular pigmentation</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Optic neuritis</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Roth spots</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>------------------</td>
<td>----</td>
<td>----</td>
</tr>
<tr>
<td>Venous tortuosity</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>
Table 3-10: Distribution of toilet type used by PEM children

<table>
<thead>
<tr>
<th>Toilet type</th>
<th>No.</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Flush toilet inside house</td>
<td>7</td>
<td>9</td>
</tr>
<tr>
<td>Bucket inside house</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Bore hole inside house</td>
<td>48</td>
<td>64</td>
</tr>
<tr>
<td>Pit latrine outside house</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>Hole on the ground outside house</td>
<td>18</td>
<td>24</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>75</strong></td>
<td><strong>100</strong></td>
</tr>
</tbody>
</table>
Table 3-11: Distribution of water supply to patients of PEM children

<table>
<thead>
<tr>
<th>Water supply</th>
<th>No.</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tap in the house</td>
<td>14</td>
<td>19</td>
</tr>
<tr>
<td>Public tap</td>
<td>9</td>
<td>12</td>
</tr>
<tr>
<td>Well</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>River</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>Buy in barrels</td>
<td>45</td>
<td>60</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>75</strong></td>
<td><strong>100</strong></td>
</tr>
</tbody>
</table>
DISCUSSION

The majority of children included in the study who had severe malnutrition were below the age of 32 months (67 patients “89.33%”) (Table 3-1). Other studies were comparatively similar. This is because infants, compared to older children, are rapidly growing with high energy requirements and can develop wasting in a relatively short period of time, secondary,
for cultures and social reasons, infants and younger children are more likely to be subjected to inadequate feeding practices and low nutritive value foods. The effects of this diet, early weaning and recurrent infections increases the risk for protein energy malnutrition in this age group.\(^{(3)}\)

The study population was found to be distributed evenly according to sex, 36 males (48%) and 39(52%) females since the disease has no sex linkage (Fig. 3.1).

Discharge and itching were the main presenting complain of the study sample. These may be due to decrease immunity which is expected in malnourished children and bad hygienic condition. These two factors (decrease immunity and bad hygiene) are strongly related to infections, e.g. conjunctivitis, trachoma and dacrocystitis.

Ocular motility was found to be full in 73 patients of the study group (Tables 3-3, 3-7). Only 2 patients had no full ocular motility, both of them their defects were due to birth trauma; one patient with incomplete ocular movement had a corneal opacity too due to the same trauma (P = 0.41). This is proved statistically (P = 0.07, insignificant) and in this study it was found that no relation between the nutritional status (marasmus, kwash, marasmic-kwash) and the ocular motility. Duane mentioned that, thiamin deficiency, which causes beriberi-manfest with progressive peripheral neuropathy involving the sensory and motor function is associated with external ophthalmoplagia affecting particularly cranial nerves III and VI, that was not found in this study and this means that there is no ocular sign of thiamin deficiency among the study group.\(^{(4)}\)

In the same tables lid chymosis, lid oedema was found in more than 45% of the study group. Seventeen of them (22.67%) had kwashiorkor, another 17 were marasmic kwash. This relation between the nutritional status of the patients and the presence of lid oedema and chemosis was found to be strongly statistically significant (P= 0.001) confirming what was found in the literature.\(^{(4)}\)

Referring also to tables 3-3 and 3-7, conjunctivitis was found in 35 patients (46.67) and dacrocystitis in one patient (1.33%). This high percentage of conjunctivitis (infections) could be
due to the secondary immunodeficiency caused by malnutrition leading to predisposition to external infections.\(^{(3,19,20)}\) In spite of that the P. value was found not significant in this study (P = 0.07 and 0.45) respectively.

Vitamin-A deficiency was an expecting finding among this study group, but signs of vitamin A deficiency was detected in 3 patients only (4%), one patient with conjunctival xerosis and the other 2 patients (2.67%) with corneal xerosis (Table 5).

In 1988 Ahmed\(^{(3)}\) in a study done in K.C.T.H about ocular manifestations of vitamin-A deficiency among 213 malnourished children, the prevalence of vitamin-A deficiency was found to be as follows, conjunctival xerosis 16%, conjunctival Bitot’s spots 5.6%, corneal xerosis 3.20%, corneal ulceration 2.8%, night blindness 4.2%, corneal scars 0.9%.\(^{(4)}\) Wadhar in 1992, observed that 15.3% of children with severe PEM admitted to KCTH had clinical signs of xerophthalmia.\(^{(2)}\) Abdelgadir, 2001\(^{(2)}\) found 4.3% of children with severe malnutrition had clinical xerophthalmia, when comparing these studies we notice that there is a reduction in vitamin-A deficiency among malnourished children, this could be attributed to the effort of the national policy of giving vitamin-A supplementation during Poliomyelitis Irradication Campaigns.

Retinal haemorrhages was found to be the only posterior segment finding in the study, it occurred in 3 patients (Table 3-6). Retinal haemorrages can occur in anaemia,\(^{(23)}\) vitamin C deficiency\(^{(3)}\) and many other diseases. It was found to be insignificasntly related to the nutritional status (P = 0.78).

Mother education and vaccination were suggested to be risk factors for eye disease among malnourished children, both were found, statistically insignificantly related to the ocular findings (P >0.05) except in dacrocystitis where the p. value was <0.05.

From table 4 the prevalence of trachoma was found to be 14.67% (11 patients), this percentage is considered to be high with the small number of the study sample. A study was done in Nile State (Sudan) by Elfatih (1999), who found active trachoma in 60% of preschool
children. Prevalence of trachoma in this study was found in agreement with Elfatih study to some extent and may be confirmed if the study sample is increased in number.\textsuperscript{(28)}

As mentioned in the literature the active form of trachoma (TF, TI) was most common in young children,\textsuperscript{(4,19,23)} TF and TI were the only stages of trachoma found in the study group (Table 4).

Trachoma is prevalent throughout most of the developing parts of the world and appears to be associated with such environmental factors as poverty, overcrowding and lack of sanitation and inadequate hygiene.\textsuperscript{(4,19,29)}

The high prevalence of trachoma among the study group may be explained by poor hygienic condition in more than 60% of the study group (Tables 3-10, 3-11). Figures 3-IV and 3-V were designed to show the relation between prevalence of trachoma and the toilet type and water supply.

Trachoma was not found to had a direct relation with the nutritional status (P= 0.1) and this might be due to the fact that trachoma is more related to the poor hygienic environmental conditions rather than the nutritional status.
CONCLUSION

The present study with its limitations has highlighted the following:

1- The majority of children included in the study were below the age of 32 months (67 patients –89.33%). The disease has no sex linkage. Lid chymosis and lid oedema was found in more than 45% of the study group; this was found to be related to the nutritional status. Statistically significant.

2- Conjunctivitis was a common finding and may be due to secondary immunodeficiency.

3- Vit. A deficiency signs was detected only in 4% of the study group. This could be attributed to vit. A supplementation during poliomyelitis eradication campaigns.

4- Retinal haemorrhage was found the only posterior segment finding occurred in 4%, insignificantly related to the nutritional status.

5- Mother education and vaccination were found statistically insignificantly related to the ocular finding.

6- High prevalence of trachoma may be explained by poor hygienic conditions of the study group.

RECOMMENDATIONS

The following are recommended:

- In Sudanese patients the ocular manifestations of severe malnutrition are notable and must be recognized by the physician.

- Training of health workers at the primary health care level and health centers on:
a- Counseling mothers on feeding, growth monitoring, the preventable eye blinding diseases and hygiene during the vaccination sessions and early referral of children with eye signs to hospitals.

b- Further studied in different aspects of eye diseases among malnourished children are needed.

REFERENCES


