UNIT 15 MAJOR DEFICIENCY DISEASES — I: PROTEIN ENERGY MALNUTRITION AND XEROPHTHALMIA

Structure

15.1 INTRODUCTION

With your understanding of the basic concepts in nutrition, you would have got a good idea about the nutrients and their role in our body. What would happen if the diet is inadequate and/or lacking in essential nutrients? Certainly, the individual would be prone to nutrient deficiency disorders. What are the major nutrient deficiency disorders prevalent in our country? Which section of the population is more prone to these diseases?

You will learn about these issues in this unit.

You are already aware that the first few years of life are crucial from growth and development point of view. Nutrition plays on important role. For instance, the body requires energy to carry out the different activities, proteins are necessary for body-building and repair, Vitamin A helps maintain normal vision etc. What would happen when there is deficiency of these nutrients? The deficiency of these basic nutrients lead to malnutrition. Malnutrition is the single largest contributor to the high rate of infant and child mortality and morbidity in India. In this unit our focus is on two nutrient deficiencies — protein energy malnutrition and Vitamin A deficiency disorder i.e. Xerophthalmia.

What are the major features, causes, clinical features of these two widespread disorders? How can we prevent them? What treatment can be prescribed? These are some of the aspects which are discussed in this unit.

Objectives

After studying this unit, you will be able to:

- describe the nature and causes of PEM and Xerophthalmia,
- enumerate the clinical features of PEM and Xerophthalmia, and
- discuss the treatment and prevention of these disorders

15.2 PROTEIN ENERGY MALNUTRITION

Protein energy malnutrition (PEM) is widely prevalent among young children (0-6 years) but is also observed as starvation in adolescents and adults, mostly lactating women, especially during periods of famine or other emergencies. PEM has serious consequences for the health of individuals, particularly, children and can even result in death.

Let us first define PEM. *PEM can be defined as a range of pathological conditions arising from a deficiency of protein and energy, and is commonly associated with*

infections. What kind of adverse changes take place in the body as a result of PEM? The adverse changes which are externally noticeable are referred to as clinical features about which we will read as we go through this section.

In Unit 2 of Block 1, you may recall reading about signs of good health related to different body parts like the eyes, the skin, the teeth etc. Many diseases result in adverse changes in the appearance and functions of one or more body parts. For example, a healthy person has clear eyes. But in a person with severe vitamin A deficiency, eyes lose their clarity and become muddy or cloudy. Similarly, a child suffering from PEM is shorter than other children of the same age. Such changes in appearance relating to the body as a whole or its parts are referred to as clinical features of a disease. The clinical features can be easily detected by trained individuals. Let us now study about the clinical features of PEM.

Clinical features of PEM

PEM is a condition characterized chiefly by the following two forms:

a) Marasmus

b) Kwashiorkor

However, there are also children who show some of the characteristic signs of both marasmus and kwashiorkor. Such children are said to suffer from *Marasmic Kwashiorkor*, Then there are children whose heights and weights are considerably below that of healthy children of the same age. These children may not show any typical clinical signs of either kwashiorkor or marasmus, and as such they are placed in the category of *subclinical forms of PEM* which forms a large proportion of the disease in the community.

What is the subclinical form of PEM?

Subclinical forms of a disease, refer to the condition where external medical examination may not show any signs of the disease. However, when we examine in detail — by body measurements like weight or examination of blood — we can detect changes. In the case of PEM, we can see a large number of subclinical forms. Kwashiorkor and marasmus are only the tip of an iceberg. In our country only 2-3 children out of a hundred, in the age group of 1-5 years, exhibit these clinical forms of PEM. However, many more subclinical cases of PEM — which cannot be easily detected by simple clinical examination, are widely prevalent in the community. For each case of kwashiorkor or marasmus, there may usually be 10 to 15 subclinical cases of PEM. For prevention of PEM, we have to take steps by identifying such cases so that prompt treatment or rehabilitation (helping child to recover his normal health) can be provided.

In all the forms of PEM, remember growth failure or low body weight is a common sign. So then, how are these forms different from each other? The description below presents a clear picture of the different forms of PEM and lists signs and symptoms specific to each form, which will help us identify individuals suffering from different forms of PEM. We begin our study by identifying signs and symptoms of marasmus.

A) How to identify a child suffering from Marasmus?

Marasmus is a condition characterized by very low body weight for age, loss of subcutaneous fat (fat under the skin), gross muscle wasting. It is observed more frequently in infants and very young children, Marasmus is usually due to very severe undernutrition i.e., a diet that is inadequate, extremely low in both proteins and calories.

Some common clinical features of marasmus include:

i) *Muscle Wasting*: The characteristic sign of marasmus is the extensive wasting of muscle with little or no fat under the skin. We use the term wasting to mean emaciation or thinness of the body. The limbs become very prominent. Because of the absence of fat, the skin develops a number of folds, particularly on the buttocks. *The child with marasmus, thus, can be described as all skin and bones.* You can see this clearly in Figure 15.1

ii) *Failure to thrive*: There is failure to thrive and the child suffering from marasmus usually is irritable and fretful. In fact, the child is often so week that the cry of the child cannot be heard.

iii) *Growth failure*: Failure to grow is another important feature of the disease. The children often weigh about 50 per cent less than children with normal weight for their age. For example, a healthy normal one year old child weighs about 10 kg, whereas, a marasmic child would weigh only about 5 to 6 kg.

In addition to these clinical features, watery diarrhoea, associated often with dehydration (loss of fluids) is usually present. The child may also have other deficiencies, particularly, vitamin A deficiency (details of which are given in Section 15.3 of this unit).

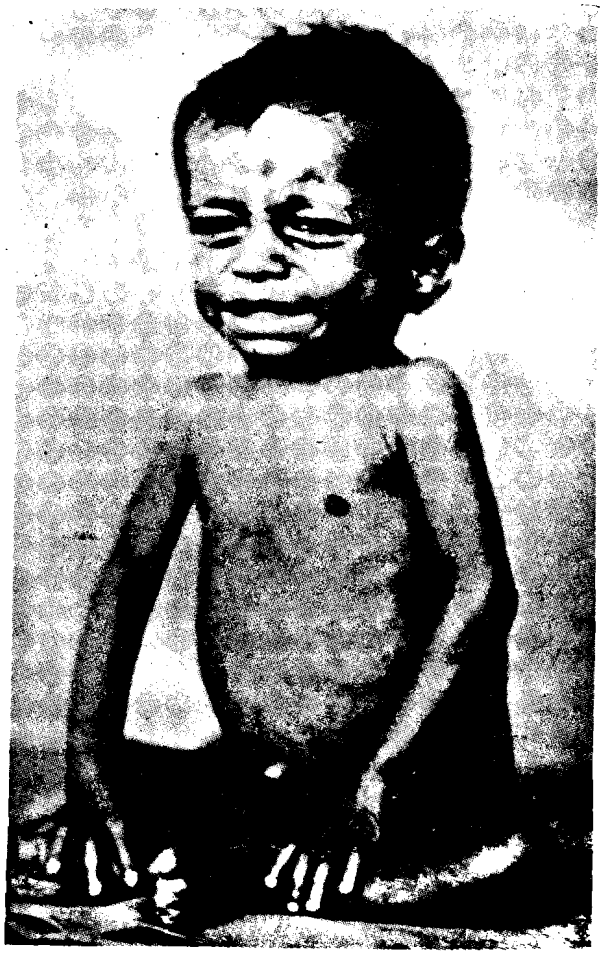

Fig. 15.1: Child with Marasmus (Photo Courtesy: National Institute of Nutrition, Hyderabad)

B) How to identify a child suffering from Kwashiorkor?

Kwashiorkor, on the other hand, is a condition characterized by oedema (excessive accumulation of fluid in the intercellular spaces of tissue) and very low body weight for age. The syndrome is most frequently observed in children age 1-3 years and is precipitated by an infection or more commonly by a series of infections. The main underlying cause of the disease, nevertheless is dietary deficiency i.e., a diet which is low in protein, but which contains calories.

Some common *clinical features* of Kwashiorkor include:

i) *Oedema*: Oedema is the excessive accumulation of fluid in the intercellular spaces of the tissues. Oedema is usually observed on the lower limbs, but it may also be distributed all over the body including the face. But how can we detect oedema? We can detect oedema by pressing the skin over the shin of the leg with the fingers. Because of accumulation of fluid under the skin, when you press there will be a depression at the place where the pressure is applied.

ii) *Failure of growth*: Growth failure is an early sign and we can notice this by taking body weight. Children with kwashiorkor weigh only about 60 per cent of the weight of normal children for their age. For example, a three year old healthy normal boy weighs about 13.5 kgs, whereas, another boy of same age but suffering from kwashiorkor may only weigh 60 per cent of 13.5 kg i.e. about 8 kg. In other words, they are very much lighter than heathy normal children of their age.

iii) *Irritability*: The child suffering from kwashiorkor is generally irritable and has no interest in his/her surroundings.

iv) *Skin changes*: In addition to the above manifestations, there may be characteristic skin changes. The skin becomes thick and appears as though it has been varnished. The skin of the child may peel off easily leaving behind cracks or sores.

v) *Hair Changes*: The hair may become sparse and can be easily pulled off. The hair usually loses its black colour and appears reddish brown.

vi) *Moon Face*: The face of the child suffering from kwashiorkor may appear puffy with the cheeks sagging. This sign is normally known as moon face.

Fig. 15.2 shows some of the clinical features like oedema, moon face and skin changes clearly.

Fig. 15.2: Child with kwashiorkor (Photo Courtesy: National Institute of Nutrition, Hyderabad)

vii) *Associated deficiencies*: The children may have signs of other deficiencies like those of vitamin A and B-complex deficiencies. What are these signs and symptoms? You will learn about these deficiencies in the subsequent units of this block.

viii) *Associated diseases*: The child suffering from Kwashiorkor is often brought to the hospital with watery diarrhoea (frequent loose motions) or severe respiratory infection (cough). The child is also prone to measles, a childhood disease, which is characterized by skin rash and fever.

What do the study of the clinical features of kwashiorkor and marasmus, reveal? Yes, Growth failure is characteristic of both these conditions. However, it is much more pronounced in marasmus. Can you then identify what exactly is the difference between these two conditions? Make a checklist and tally your responses with principal features of PEM given in Table 15.1.

Table 15.1: Principal features of PEM

Features	Marasmus	Kwashiorkor
Essential features	• extensive muscle wasting (prominent ribs, skin)	• oedema
	• total loss of subcutaneous fat	• low body weight for age
	• growth retardation in terms of body weight (low body weight for age)	• mental changes
Variable	• diarrhoea (often)	• poor appetite
		• skin changes (scars, cracks, peeling of the skin)
		• hair changes (dyspigmentation)
		• moon face (often)
		• diarrhoea (often)
		• liver enlargement

C) How to identify Marasmic kwashiorkor in children?

In India, particularly among poorer families, we also come across patients who present a mixture of both kwashiorkor and marasmus. The children *are not only extremely wasted like in marasmus but also have signs of kwashiorkor i.e. have swelling of feet (oedema).*

D) How to identify subclinical forms of kwashiorkor?

Body weights is a good indicator of the subclinical forms of PEM.

A child's body weight is compared with that of a well fed, healthy normal child of the same age. The weights of normal children used for such a comparison are known as reference standards. These tables of normal weights and heights of healthy normal children provide weights and heights according to age. In India and elsewhere, the reference standards developed by the National Centre for Health Statistics (NCHS), USA are extensively used. The reason is that the growth pattern of healthy Indian children (atleast until adolescence) is comparable with that of American children.

Table 15.2 gives the weights of healthy American children which are recommended to be used as standards.

Table 15.2: Average Weights of Healthy American Children (NCHS)*

Age (months)	Weight (Kg.)	
	Boys	Girls
Birth	3.3	3.2
6	7.8	7.2
12	10.2	9.5
18	11.5	10.8
24	12.3	11.8
30	13.5	13.0
36	14.6.	14.1
42	15.7	15.1
48	16.7	16.0
54	17.7	16.8
60	18.7	17.7
66	19.7	18.6
72	20.7	19.5

* National Centre for Health Statistics

By comparing the weights of children with those of healthy normals we can classify the children into different forms of malnutrition. The Indian Academy of Pediatrics (IAP) has recommended the following classification (Table 15.3) for detecting different forms of PEM using weight for age. You will learn about weight for age as a measurement to assess nutritional status in Unit 20, Block 6.

Table 15.3: Simple Classification of PEM

STATUS	CRITERIA
Normal	More than 80% NCHS weight for age
Mild Malnutrition	70-80% NCHS weight for age
Moderate Malnutrition	60-70% NCHS weight for age
Moderately Severe Malnutrition	50-60% NCHS weight for age
Severe Malnutrition	Less than 50% NCHS weight for age

Let us understand the classification, using an example.

Consider a child of one year age. A normal healthy male child of this age should have a weight of 10.2 kg as indicated in Table 15.2. Children weighing more than 80 per cent of healthy children are considered as normal as per the IAP classification. Eighty per cent of 10.2 kg is approximately 8.2 kg. So a child weighing more than 8.2 kg is a normal child. Similarly, you can calculate 70 per cent of the weight of 1 year old normal, healthy children. In this case it is equivalent to 7.1 kg. So, children with weights between 7.1 and 8.2 kg are considered as suffering from mild malnutrition. You can calculate the other grades in a similar way.

You may be wondering what is the use of such a classification. Information given in Highlight 1 will draw your attention to this crucial aspect.

HIGHLIGHT 1 **Supplementary Feeding Programmes**

Malnutrition is the major problem facing the country. Children suffering from malnutrition need special attention. Those in moderate and mild degree of malnutrition require additional food in order to bring their weight to normal, whereas, those children suffering from a severe degree of malnutrition, definitely need special attention by a health functionary as they usually suffer from diarrhoea, respiratory infections and other diseases. The crucial question is how do we detect these cases of malnutrition. We can make use of the classification given in Table 15.3 to identify cases of malnutrition. From this information we can then select children to participate in feeding programmes. Generally, those children with moderate and severe malnutrition, based on weight for age, are included in the supplementary feeding programmes. What are these supplementary feeding programmes? Supplementary feeding programmes are simple nutrition intervention programmes initiated by the government to meet the nutrient needs of the vulnerable sections of the society (specially children and pregnant/lactating women). You will read about them in Unit 18 of Block 5. The main objectives of the feeding programmes are:

- to supplement the diets of the weaker sections of the community to correct undernutrition, and
- to educate the community for combating and preventing malnutrition.

From our discussion so far we have got an idea of how to identify the different forms of PEM. The next question which comes to our mind is what are the causes of this widespread disorder? The discussion which follows after the Check Your Progress Exercise presents a detailed review of some of the causes of PEM.

Check Your Progress Exercise 1

1) What are the differences in the clinical features of kwashiorkor and marasmus?

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2) Fill in the blanks.

a) A male child of one year weighs 4.7 kg as against the normal of 9.5 kg. The child is suffering from ..... form of PEM.

b) A child who has extensive wasting of muscle is suffering from .....

c) In the case of PEM, subclinical forms can be identified by using .....

d) ..... is the condition characterized by sagging cheeks and puffy face.

e) ..... is the condition, where external medical examination may not show any sign of the disease.

What are the causative factors of PEM?

Some of the causes of PEM are:

a) *Poverty:* PEM occurs in poor Indian communities. It is commonly seen in families of landless agricultural labourers, backward communities and nomadic tribes. These

communities are poor, illiterate and without any regular earnings, therefore, they are unable to provide enough food for their large families. Children living in urban slums are also at risk.

b) *Maternal malnutrition:* Do you recall the discussion we had in Unit 9 of Block 3 on nutritional status of the mother and its impact on pregnancy? We learnt that the nutritional status of the mother determines the state of nutrition of the child to be born. If the nutritional status of the mother is poor, the chances of the offspring being malnourished are higher. Maternal malnutrition, results in low birth weight of offspring. Infants with birth weight lower than 2500 gms (2.5 kg) are considered as low birth weight babies. Children who develop PEM often begin life with a low birth weight.

c) *Infections and poor hygiene:* Generally, kwashiorkor follows attacks of diarrhoea (frequent loose motions) or an attack of measles. In the urban slums, in particular, artificial feeding with commercial milk foods is common. The mothers may follow unsound and unhygienic methods of feeding the child. Feeding bottles may not be properly sterilized. Flies may be allowed to sit on the nipple of the feeding bottle. This may lead to frequent diarrhoea and lead to marasmus. Hence, the importance of good hygiene is not realized leading to ill-health and malnutrition.

d) *Ignorance:* Both the forms of PEM occur as a result of ignorance on part of the mother. You have read about the close relationship between malnutrition and infection in Unit 2 (Block 1) of this course. The mother, due to ignorance delays the introduction of supplementary food even upto the age of 1 year. This has serious consequences because mother's milk alone is not enough for the child By the age of 6 months, the infant should be given supplementary foods in addition to breast milk. Further, in case when the child is suffering from infections such as diarrhoea, measles and common fevers the mother restricts the diet of the child. This practice is not good since such a dietary restriction leads to PEM in children who are already underfed.

e) *Wrong child feeding practices:* You have learnt that supplementary foods in addition to breast milk are introduced quite late. The child is usually given the same diet taken by adults and very often follow the same meal pattern. The typical Indian diet is based on cereals and is quite bulky for a small child. This would mean that the child can consume only smaller amounts of the food at one time. But you may recall reading in Unit 2 of Block 3 that the child need frequent meals atleast four to five times a day to meet the daily requirement of nutrients. Traditionally an Indian child is fed three times a day. As a result, the child does not get adequate food — that is enough energy, protein — which is the major cause of PEM in India.

Now that you are aware of the causative factors of PEM, let us move on to discuss the prevention and treatment of this disorder.

What is the treatment for PEM?

You know PEM is caused due to lack of food, in other words, a deficiency of energy and protein in the diet. So one of the major objectives of the treatment is to feed the child energy and protein-rich foods, so that the dietary requirements are met and there is adequate weight gain. This treatment can be very well done at home with judicious selection of energy and protein-rich foods. However, the children with severe malnutrition most often, require hospitalization since they may also have associated infections like severe gastroenteritis (infection of digestive tract) and severe respiratory infections. If you come across such a case, ask the mother to take the child to a health centre immediately. In the initial stages it will be difficult to feed such children. They may require feeding through a rubber tube passed through the nose into the stomach till they are able to take the food by mouth. Initially only high calorie liquid foods are given. Some of these cases can also be treated at home with proper supervision, if they do not have any severe infections like diarrhoea etc. The main principle in the treatment of the severe forms of PEM is to provide adequate energy and protein through dietary means. This can be provided through the foodstuffs like cereals, pulses, nuts and sugar/jaggery usually consumed by the community at their homes. Addition of milk is not compulsory but, if added, will improve the quality of the diet. Remember, that there is no need to give high protein commercial preparation. A nutritious, low cost preparation used in treatment of PEM — Hyderabad Mix — is discussed in Highlight 2.

HIGHLIGHT 2 **Hyderabad Mix**

A preparation based on local foods which was developed by the National Institute of Nutrition, Hyderabad and successfully used in the treatment of kwashiorkor and marasmus is the Hyderabad Mix. This Hyderabad mix, which is not a commercial food, can be prepared even in rural households belonging to poorer families. It consists of roasted wheat (40 grams), roasted bengal gram dal (15 grams), roasted groundnut (10 grams) and sugar/jaggery (30 grams). We can prepare ladoos, or sweet kheer with this mix or it can be cooked with milk to improve the taste and quality.

In about 4-6 weeks, the children with kwashiorkor recover fully with dietary treatment though cases of marasmus take longer periods. It is important to remember that during the first week the child requires persuasive efforts and coaxing for feeding. After this, once the child recovers the appetite, feeding becomes much easier. In young children of 6-24 months, who also have a problem in swallowing solid food, one can make a thin gruel of the Hyderabad mix by adding a few grams (1/2 teaspoon) of ARF powder (germinated wheat flour powder). Addition of ARF to the gruel would help make the gruel thin but at the same time retain its nutritive value. The child with PEM can easily drink this gruel.

Simultaneously other deficiencies like those of vitamin A and B-complex should also be treated with vitamin A capsules and tablets of B-complex. In addition, associated infections should be controlled with appropriate medicines. The mother should be educated to continue feeding additional food by increasing the quantities of the home diet even after discharge from the hospital (if the child had been admitted) so that the child may not get PEM once again.

Mild and moderate cases of PEM can be treated at home by giving energy and protein rich diets as mentioned earlier. They do not require hospitalization.

How to prevent PEM?

You know that "prevention is better than cure". A few measures, which when followed will prevent PEM are listed in the following discussion:

i) Prevention of PEM begins with the mother of the child. You have learnt that children with low birth weights often develop PEM. The main reason for low birth weight is maternal malnutrition. It is, therefore, important to ensure that a pregnant woman consumes extra food to meet the additional needs of pregnancy. A simple thumb rule is to ensure that pregnant women consume additional amounts of foods — equal to one normal meal — every day. In the case of poorer families, supplementary food (food in addition to home diet) can be given to the pregnant women during the last three months of pregnancy under government's feeding programmes. These steps would help in improving the birth weight of the children.

ii) Mother's milk is the best food for an infant. Lactating mothers should be encouraged to breast feed their children as long as possible. By the age of 4-5 months, however, mother's milk alone is not adequate for the child. Supplementary food should be provided to the children, in addition to breast milk. These can be cereal—pulse and nut mixes (for eg. wheat, bengal gram and groundnut mix) and can be prepared at home by the mother.

iii) Children should be fed 4-5 times a day. We have already learnt that cereal-based Indian diets are quite bulky and hence the child cannot eat much at one time. Unless the child is fed frequently it cannot meet the energy and protein requirements. Hence, small but frequent meal is a good infant feeding practice.

iv) Infections like diarrhoea and respiratory infections increase the risk of PEM. Prompt treatment of these infections would, therefore, help to prevent PEM. In addition, during diarrhoea and any other infection, food should not be restricted. The child should be fed as usual.

v) Protection of children against infections diseases like tuberculosis, measles, or whooping cough by immunization is another important aspect in the prevention of PEM. Under the universal immunization programme all the infants receive immunization against all these diseases which are important contributory factors in child malnutrition. Mothers should be educated to avail of these services.

Check Your Progress Exercise 2

1) List any three measures which you would like to adopt to prevent PEM in a village community.

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2) List the main causes of PEM in infants.

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15.3 XEROPHTHALMIA

Vitamin A deficiency is a serious public health problem in India. Xerophthalmia, refers to the eye manifestations (signs) arising due to vitamin A deficiency. Blindness, resulting due to xerophthalmia, is a major health problem. Although vitamin A deficiency may become apparent at all ages, the preschool child is the most frequent victim of this debilitating disorder. According to rough estimates thirty thousand to forty thousand children may loose their eyesight due to vitamin A deficiency in India. (Source: Menon K and Vijayraghavan K. Sequence of severe Xerophthalmia. A follow-up study. Am J. Clin. Nutr. 1980).

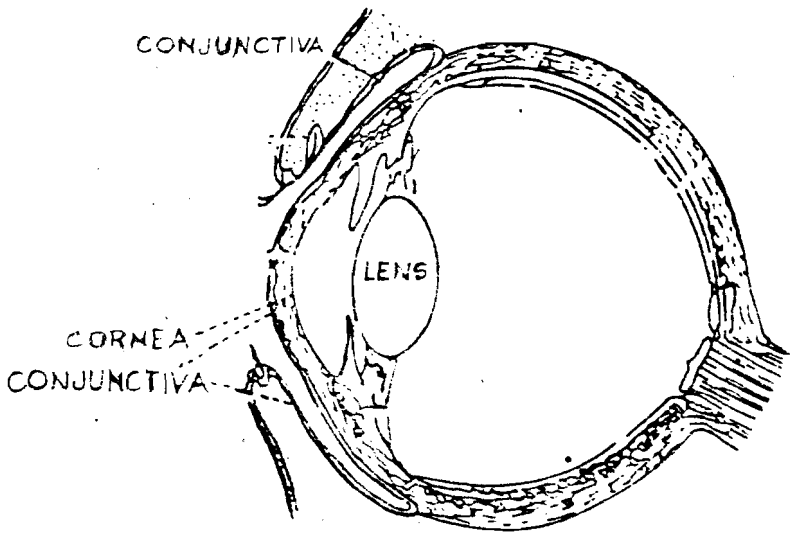
What are the signs and symptoms of this disorder? On reading through this section you will be introduced to the various eye changes (clinical features) common to Vitamin A deficiency.

Clinical features of Xerophthalmia

The signs and symptoms of xerophthalmia pertain to changes in the eye. Do you recall the figure of the eye given in Unit 6 of Block 2? The figure represented the structure of an eye. Once again for your convenience the figure is given here. In this figure you can see the various part of the eye. It is the conjunctiva (thin transparent membrance that covers the cornea and lines the inside of the eyelid) and the cornea (the anterior, transparent portion of the outermost layer of the eye) which are most often affected by the deficiency.

Let us now look at the signs and symptoms of xerophthalmia.

a) *Night Blindness:* One of the earliest manifestations of xerophthalmia is night blindness. Individuals suffering from night blindness cannot see in dim light or around dusk. The child will be unable to see even the meal plate kept in front of him/her in dim light. This condition is known by different names in different regions. For example, in the rural areas of the North (Hindi belt) this condition is commonly known as rathoundi.



b) *Conjunctival Xerosis:* Xerosis in Greek means dryness. Conjunctival xerosis, therefore, means dryness of the conjunctiva. In the normal eyes, the membrane covering the white portion of the eye (i.e. conjunctiva) is bright, white and moist. In the case of xerophthalmia, it becomes discoloured (muddy coloured), dry and loses its brightness. This is known as conjunctival xerosis. Even when there are tears in the eyes the conjunctiva cannot be wetted.

c) *Bitot spots:* In addition to Xerosis dry, foamy, triangular spots may appear on the conjunctiva. These are known as Bitot's spots (Figure 15.3). Usually these are more common on the temporal side (towards the ear) of the eye rather than the nasal (towards the nose) side. In our country there is a practice among girls to apply 'kajal' (eyetex kohl) to their eyes. In such cases, Bitot spots also take up the black colour of the 'kajal' and can be seen even from a distance. Though conjunctival changes in xerophthalmia do not lead to blindness they should be considered as warning signs. If neglected, these changes may progress affecting the cornea and may lead to irreversible blindness.

d) *Corneal xerosis:* As in the case of the conjunctiva, the normal cornea (the anterior, transparent portion of the outermost layer of the eye) is moist and shining. When vitamin A deficiency becomes severe, the cornea becomes dry and dull and appears like broken ground glass. This condition should be treated as an emergency. If it is not treated immediately with vitamin A, the child can develop ulcers (sores) in the cornea. Corneal ulcers (corneal sores) when healed leave white scars known as leucoma. Such white scars on the black of the eye can interfere with normal vision.

Fig. 15.3: Child with Bitot's spots (Photo courtesy: National Institute of Nutrition, Hyderabad)

e) *Keratomalacia:* The most dangerous form of xerophthalmia is known as keratomalacia. In this condition, the cornea becomes very soft and raw and easily infected. It leads to destruction of the eye. In other words, the eye get completely melted and destroyed (Figure 15.4). This condition inevitably leads to irreversible blindness. Generally, this condition is seen in both the eyes and is common in children between the ages of 1-5 years. About 95-96 per cent of the children with keratomalacia also have either kwashiorkor or marasmus. In addition, the prevalence of infection is also high in these children. Subsequently, sixty to sixty five per cent of these children die.

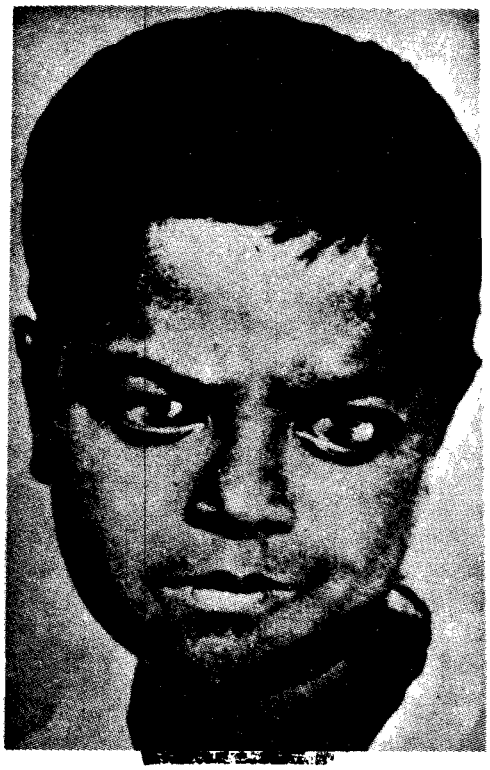


Fig. 15.4: A child who became blind due to Keratomalacia (Photo courtesy: National Institute of Nutrition, Hyderabad)

The study of this section would have given you an idea of how to identify signs/ symptoms of vitamin A deficiency. But, what is equally important for us is to know is, what are the causative factors of this disorder? Let us now learn about them.

What are the causative factors of Xerophthalmia?

Xerophthalmia is common in the families of low socio-economic group living in rural areas and urban slums. In India, the disease is more common among the poorer sections of the community. Vitamin A deficiency may become apparent at all ages. But, the most common, predominantly nutritional, variety occurs in the third and fourth years of life, at least in countries where breast feeding is prolonged. The younger the child, the more serious the manifestations and the greater the mortality rate. The cornea is rarely affected in children beyond the age of five years. In fact, the prevalence of corneal xerophthalmia is maximum between the ages of 1 and 3 years. The disease is relatively more frequent among males. The main causes being:

a) *Maternal Malnutrition:* Indian children of very poor rural families are born with low liver stores of vitamin A because their mothers are also deficient in vitamin A. The women during pregnancy continue to consume very low amounts of vitamin A either due to poverty or ignorance. As a result, the children born to such women have low vitamin A reserves in the body. You have already learnt earlier in Unit 6, Block 2 that vitamin A is fat-soluble and can be stored in the body for longer periods.

b) *Dietary inadequacy of Vitamin A:* The primary cause of xerophthalmia is dietary inadequacy of vitamin A. In the villages and urban slums, among the low income groups, the intake of vitamin A is less than a quarter of the Recommended Dietary Intakes (RDI). As long as the child is breast fed, the vitamin A status of the infants is apparently adequate because the infant gets reasonable amounts of vitamin A through breast milk. Once the child is taken off the breast, the child is put on the family diet which is deficient in vitamin A. Due to inadequate consumption of vitamin A, the child develops vitamin A deficiency manifesting as xerophthalmia.

c) *Infections and Infestations:* Diarrhoea and respiratory infections and worm infestations like round-worm disease are very common in young children. These are known to decrease the absorption of vitamin A and lead to its deficiency.

Measles, one of the childhood infections, is another important cause of xerophthalmia leading particularly to corneal sores and blindness.

Few of the most common causes of xerophthalmia have been discussed above. What preventive measures and treatment can be prescribed to overcome this disorder is the next point of discussion.

What is the treatment for Vitamin A deficiency?

For the purpose of treatment we have to first distinguish between milder forms and severe forms of vitamin A deficiency. You have already learnt that the involvement of the cornea should be considered as the most severe form, since, it can lead to blindness. These should be treated without any delay. Such cases need to be given high doses of a vitamin A as injections as well as by mouth.

Children with night blindness, conjunctival xerosis and Bitot spots are treated with a massive oral (by mouth) dose of vitamin A.

How to prevent Vitamin A deficiency?

A few measures to prevent vitamin A deficiency are listed below:

i) *Consume Vitamin A rich diet:* We know that xerophthalmia is primarily due to dietary inadequacy of vitamin A. Therefore, the most rational method of prevention of vitamin A deficiency is to consume foods rich in vitamin A regularly. Inexpensive foods like green leafy vegetables (palak, amaranth, etc.), yellow vegetables (yellow pumpkin and carrots) and fruits (papaya and mango) are good sources of beta carotene (precursor of vitamin A). Consumption of as little as 40 grams of green leafy vegetables daily is enough to maintain the normal vitamin A status in children. Feeding milk, cheese, curd, butter, eggs, liver, fishes, red palm oil — the other rich sources of Vitamin A — should be emphasized. Improvement of Indian diets, however, requires extensive and continuous nutrition education programmes. Ensuring regular and adequate intake of Vitamin A by preschool children, pregnant and lactating women should be crucial to prevent Vitamin A deficiency.

ii) *Periodic administration of Vitamin A:* Blindness due to xerophthalmia is a serious problem and requires the most urgent measures. We have already learnt that vitamin A can be stored in the body for prolonged periods. It is possible to build up sufficient vitamin A stores in a child by giving large doses of vitamin A periodically. Using this principle, a national programme for prevention of blindness due to xerophthalmia has been developed by the National Institute of Nutrition (NIN) and is operated by the Government of India in the different States of the country. Under the programme, the children between the ages of one and five years are given a massive oral dose of vitamin A once every six months. The distribution of vitamin A is carried out by the village level health workers like multipurpose health workers of the State Governments. Such a programme, when properly carried out, can reduce incidence of blindness due to xerophthalmia in 80 per cent of young children. Simultaneous nutrition education is also important for the success of the programme. You will learn more about this programme in Unit 18 of Block 6.

Check Your Progress Exercise 3

1) Fill in the blanks:

   a) Xerophthalmia is due to the deficiency of ..... in the diet.

   b) Conjunctival xerosis means ..... of conjunctiva.

   c) The most dangerous consequence of xerophthalmia is irreversible

   .....

   d) Dry, foamy, triangular spots appearing on the conjunctiva are called

   .....

2) The clinical features of xerophthalmia relating to the cornea are :

   .....

   .....

   .....

3) List the main causes of xerophthalmia in India.

   .....

   .....

   .....

4) What advice will you give to a mother of a preschool child in a village to prevent xerophthalmia?

   .....

   .....

   .....

15.4 SUMMING UP

In this unit we studied about the two major nutritional deficiency disorders — PEM and Xerophthalmia.

PEM is very common in our country and kwashiorkor and marasmus are the severe forms. Oedema is present only in kwashiorkor and there is extreme muscle wasting in marasmus. PEM is due to dietary deficiency of energy and protein. Maternal ignorance, childhood infections, and low birth weight are the contributory causes. These can be treated by consuming foods prepared from cereal, pulse, nuts, jaggery or sugar and other such energy, protein-rich foods. Improvement of the nutritional status of pregnant women, breast feeding the baby, supplementation at about 6 months of age, immunization and prompt treatment of minor diseases like respiratory infections and diarrhoea can help in the prevention of PEM.

Xerophthalmia is due to vitamin A deficiency. It leads to irreversible blindness particulary in young children. The clinical manifestations are seen in the eyes. These are night blindness, conjunctival xerosis, Bitot spots, corneal xerosis, corneal ulcer and keratomalacia. Dietary inadequacy, infections, infestations and ignorance on the part of mothers are the contributory factors for vitamin A deficiency. It can be easily prevented by consumption of inexpensive Vitamin A-rich foods like green leafy vegetables, carrots, yellow pumpkin, papaya and mango. Blindness due to xerophthalmia can be prevented by administration of massive dose of vitamin A, every six months to the children between 1 and 5 years of age.

15.7 GLOSSARY

International Standards for weight : Body weights of normal and well fed children, usually obtained on American children used as the ideal standard for comparison.

Intramuscular injection : Injection of medicines into muscle for treatment of disease(s).

Malnutrition : Condition occurring due to deficient or excessive intake of nutrients.

Microgram (ug) : 1/100th of a miligram

Precursor : It refers to any substance which can be converted to a vitamin in the body. For example, carotene is the precursor of Vitamin A.

Subclinical : Disease condition which occurs before clinical signs/ symptoms of a disease occur.

15.6 ANSWERS TO CHECK YOUR PROGRESS EXERCISES

Check Your Progress Exercise 1

1)

Features	Kwashiorkor	Marasmus
Oedema	Present	Absent
Muscle wasting	Moderate	Severe
Hair changes	Present	Absent
Body weight	60% of normal	50% of normal
Moon face	Present	Absent

2) a) Severe (i.e $\frac{4.7}{9.5} \times 100 = 49.5$ which is less than 50% for age)

b) marasmus

c) body weight

d) Moon face

e) Subclinical forms

Check Your Progress Exercise 2

1) Any three of the following:

a) Improve maternal nutrition by ensuring adequate intake of energy-rich foods.

b) Breast feed the child as long as possible.

c) Introducing supplementary food in addition to breast milk by the age of 6 months.

d) Children should be fed 5-6 times a day.

e) Prompt treatment of minor ailments like diarrhoea and respiratory infection.

f) Immunization of children against diseases.

2) Poverty, Maternal malnutrition, Infections, Poor hygiene, Ignorance, Wrong feeding practices.

Check Your Progress Exercise 3

1) a) Vitamin A

   b) dryness

   c) blindness

   d) Bitot spots

2) Corneal xerosis, corneal ulceration and keratomalacia.

3) a) Maternal malnutrition.

   b) Deficiency of vitamin A in the diets

   c) Infections like diarrhoea, measles and respiratory infections

   d) Infestations like round worm disease

   e) Ignorance leading to avoidance of vitamin A-rich foods in the diet

4) • Give daily one of the inexpensive local foods like green leafy vegetables, carrots, yellow pumpkin, papaya or mango. Consuming milk, cheese, egg and fish (if eaten) the other rich sources will be useful.

   • Provide massive oral dose of Vitamin A once every six months.