UNIT 17 MAJOR DEFICIENCY DISEASES-I:
PROTEIN ENERGY MALNUTRITION
AND XEROPHTHALMIA

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17.1 INTRODUCTION

This unit will deal with two of the major deficiency diseases, namely Protein energy malnutrition (PEM) and Xerophthalmia (Vitamin A deficiency).

You learnt earlier that the human body requires energy to carry out the different activities and proteins are necessary for growth and repair. In this unit we shall learn as to what happens when there is deficiency of energy and protein in the diet. The deficiency of these two basic nutrients in our body leads to protein energy malnutrition. Similarly due to lack of Vitamin A rich foods in the diet, vitamin A deficiency or xerophthalmia results. These two deficiency disorders have very serious consequences and are major nutritional problems in India.

Which section of the population group is more vulnerable to these diseases? 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 issues 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.

17.2 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 1 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

*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.

*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 aged 1-3 years and is precipitated by an infection or more commonly by a series of infections.

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. Subclinical condition means that we do not see the clinical features of the disease. These forms of the disease can be identified only on special investigations or tests. In the case of PEM, we can detect subclinical status by measuring body weight.

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?**

Some common clinical features of marasmus include:

1. **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 ribs become very prominent. Because of the absence of fat, the skin will develop a number of folds, particularly on the buttocks. The child with marasmus, thus, can be described as skin and bones.

2. **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 weak that the cry of the child cannot even be heard.

3. **Growth failure**: Failure to grow is another important feature of the disease. The children often weigh about 50 per cent or less of normal children 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 there is usually watery diarrhoea associated often with dehydration (loss of fluids). The child may also have other deficiencies particularly, vitamin A deficiency (details of which are given in Section 17.3 of this unit).
B. How to identify a child suffering from Kwashiorkor?

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. *Remember kwashiorkor should not be diagnosed without the presence of oedema*. But how can we detect oedema? We can detect oedema by pressing the skin over the shin of the leg with your 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 healthy 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. 17.2 shows some of the clinical features like oedema, moon face and skin changes clearly.

![Figure 17.2 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 is often brought to the hospital with watery diarrhoea (frequent loose motions) or severe respiratory infection (cough). The children often will be recovering from measles, a childhood disease, which is characterized by skin rash and fever.

Our study of the clinical features of kwashiorkor and marasmus, reveal that growth failure is characteristic of both these conditions. However, it is much more pronounced in marasmus. Can you now 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 17.1.

<table>
<thead>
<tr>
<th>Table 17.1: Principal features of PEM</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Features</strong></td>
</tr>
<tr>
<td>Essential features</td>
</tr>
<tr>
<td></td>
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<tr>
<td></td>
</tr>
</tbody>
</table>
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. You have earlier learnt that these are referred to as marasmic kwashiorkor. 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?

Subclinical forms of a disease, as you know, 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 actually considered as 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. How do we diagnose these? We have already learnt that children with kwashiorkor or marasmus have very low body weights. Similarly, by taking body weights we can also identify 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) of the USA are extensively used. The reason is that the growth pattern of well-to-do Indian children (atleast until adolescence) is comparable with that of American children.

Table 17.2 gives the weights of healthy American children which are recommended to be used as standards.

<table>
<thead>
<tr>
<th>Age (Months)</th>
<th>Weight (Kg.)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Boys</td>
</tr>
<tr>
<td>Birth</td>
<td>3.3</td>
</tr>
<tr>
<td>6</td>
<td>7.8</td>
</tr>
<tr>
<td>12</td>
<td>10.2</td>
</tr>
<tr>
<td>18</td>
<td>11.5</td>
</tr>
<tr>
<td>24</td>
<td>12.3</td>
</tr>
<tr>
<td>30</td>
<td>13.5</td>
</tr>
<tr>
<td>36</td>
<td>14.6</td>
</tr>
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<td>42</td>
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<tr>
<td>54</td>
<td>17.7</td>
</tr>
<tr>
<td>60</td>
<td>18.7</td>
</tr>
<tr>
<td>66</td>
<td>19.7</td>
</tr>
<tr>
<td>72</td>
<td>20.7</td>
</tr>
</tbody>
</table>

* 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) recommended the following classification (Table 17.3) for detecting different forms of PEM using weight for age:

<table>
<thead>
<tr>
<th>STATUS</th>
<th>CRITERIA</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>More than 80% NCHS weight for age</td>
</tr>
<tr>
<td>Mild Malnutrition</td>
<td>70-80% NCHS weight for age</td>
</tr>
<tr>
<td>Moderate Malnutrition</td>
<td>60-70% NCHS weight for age</td>
</tr>
<tr>
<td>Moderately Severe Malnutrition</td>
<td>50-60% NCHS weight for age</td>
</tr>
<tr>
<td>Severe Malnutrition</td>
<td>Less than 50% NCHS weight for age</td>
</tr>
</tbody>
</table>

For example, let us 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 17.2. You know that 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 8.16 kg or 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 children. In this case it is equivalent to 7.14 or 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 malnutrition require additional food in order to bring their weight to normal, whereas, those children suffering from a severe degree of malnutrition need special attention by a health functionary as they usually suffer from diarrhea, respiratory infections and other diseases. The crucial aspect, however, is how do we detect these cases of malnutrition. We can make use of the classification given in Table 17.3 to identify cases of malnutrition. Based on 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 help meet the nutrient needs of the vulnerable sections of the society (specially children and pregnant/lactating women). You will read about them in Unit 24 of Block 6.** The main objectives of the feeding programmes are:

- to supplement the diets of the weaker sections of the community to combat undernutrition and
- to educate the community for combating and preventing malnurtion.

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 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?

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, and tribal communities without any regular earnings among others. In India, PEM is seen in backward communities of Harijans, nomadic tribes and children in urban slums. These communities are poor, illiterate and generally have large families.

b) Maternal malnutrition: Do you recall the discussion we had in Unit 8 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. A poor Indian infant starts life with a handicap. The weight at birth of such an Indian child is lower than the normal children. You are aware that a normal infant is about 3 kg at birth. Infants with birth weights lower than 2500 grams (2.5 kg) are considered as low birth weight babies. In rural areas (villages) among poorer groups for every 100 children about 30-35 have low birth weight. In well fed, normal and healthy children this is noticed only in less than ten out of 100 children. Children who develop PEM often begin life with a low birth weight. You will find a detailed discussion on maternal malnutrition in Unit 22.

c) Infections and poor hygiene: Generally, kwashiorkor follows attacks of diarrhoea (frequent loose motions) or an attack of measles. You will learn the reasons for this in Unit 20 of this block. In the urban slum areas, 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.

d) Ignorance: Both the forms of PEM occur as a result of ignorance of the mother, in addition to poverty. The mother, due to ignorance, delays the introduction of supplementary food (in addition to breast milk), even up to 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. In addition, mothers restrict the diet when the child is suffering from infections such as diarrhoea, measles and common fevers. This practice is not good since such a dietary restriction leads to PEM in children who are 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. 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 remember reading in Unit 9 of Block 3 that the child should be given frequent meals at least five to six times a day to meet the daily requirement of nutrients. However, traditionally an Indian child is fed thrice a day. As a result, the child does not get adequate food. Consequently, the child cannot get enough energy, protein which is the major cause of PEM in India. It may be mentioned here that the protein intake is, however, just a little less than what is required. It is the intake of energy which is largely deficit.

What is the treatment for PEM?

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

You know PEM is caused due to deficiency of energy and protein in the diet or in other words, due to lack of food. So one of the major objectives of the treatment is to feed the child energy and protein-rich foods, so that his 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 center where treatment for these infections is given first priority. 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. We can also treat some of these cases at the homes of the children with proper supervision, if they do not have any severe infections like diarrhea 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 usually consumed by the community at their homes like cereals, pulses, nuts and sugar/jaggery. 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 preparations. A nutritious preparation used in treatment of PEM 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 or 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 (30 grams) and sugar/jaggery (30 grams). We can prepare laddus 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 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). You have already learnt how to make the ARF powder in Unit 9 of Block 3. Addition of ARF to the gruel would help make it thin but at the same time retain its nutritive value. The child with PEM can easily drink this up.

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 antibiotics. The mother should be educated to continue feeding of additional food by increasing the quantities of the home diet even after discharge from the hospital 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”. Few measures, which when followed will prevent PEM are listed in the following discussion:

i) Prevention of PEM should start with the mother of the child. You have learnt that children with low birth weights often develop PEM. As you have already learnt, the main reason for low birth weight is maternal malnutrition i.e. the mother of the child consumes inadequate quantities of energy and protein during her pregnancy. We have to, therefore, 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 during pregnancy*. 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 weights 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 about 6 months, however, mother’s milk alone is not adequate for the child. *Supplementary food should be provided to the children by the age of six months*, 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. You could refer to Annexures 3 and 4 of Block 3 for more ideas on supplementary foods for infants and Annexure 5 for nutritious snacks for preschoolers.

iii) *Children should be fed 5-6 times a day*. We have already learnt that cereal-based Indian diets are quite bulky and unless the child is fed frequently it cannot meet the energy and protein requirements.

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 diseases like tuberculosis or measles, 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.

2) List the main causes of PEM in infants.
17.3 XEROPHTHALMIA

Xerophthalmia refers to the eye manifestations (signs) arising due to vitamin A deficiency. Blindness, resulting as a result of xerophthalmia, is an important public health problem in India. It is estimated that about a quarter of the 15 million blind persons in the country are due to xerophthalmia. Although vitamin A deficiency may become apparent at all ages, the preschool child (in the third or fourth year of life) is the most frequent victim of this debilitating disorder. What are the signs and symptoms of this disorder? On reading through the subsequent section you will be introduced to the various eye changes (clinical features) common to Vitamin A deficiency.

Clinical features of Xerophthalmia

The clinical manifestations in xerophthalmia pertain to changes in the eye. Do you recall the figure of the eye given in Unit 4 of Block 2? The figure represented the structure of an eye. Once again for your convenience this figure is given here. In this figure you can see the various parts of the eye. It is the conjunctiva and the cornea—the two parts of the eye which are most often affected by the deficiency.

Let us now look at the clinical manifestations 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* (thin transparent membrane that covers the cornea and lines the inside of the eyelid). 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.

Bitot spots: In addition to xerosis, dry foamy, triangular spots may appear on the conjunctiva. These are known as Bitot’s spots (Figure 17.3). 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’ (eyeliner 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, the 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 ground glass. This condition is called corneal xerosis which means dryness of the cornea. This condition should be treated as an emergency. If it is not treated immediately with vitamin A, the child can develop ulcers (sore) 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.

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 gets completely melted and destroyed (Figure 17.4). 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. Sixty to sixty-five per cent of these children die. In addition, the prevalence of infections is also high in these children. About 95-96 per cent of the children with keratomalacia also have either kwashiorkor or marasmus.
The study of this section would have given you an idea of how to identify clinical manifestations of vitamin A deficiency. But, what is equally important for us is to know 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 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 causes being:

a) 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). Do you recall the RDI for vitamin A for different ages? For your reference the RDIs for different ages are given below:

<table>
<thead>
<tr>
<th>Category</th>
<th>Retinol (μg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Adult Man/Woman</td>
<td>600</td>
</tr>
<tr>
<td>Lactating Woman</td>
<td>950</td>
</tr>
<tr>
<td>Infants (1 year)</td>
<td>300</td>
</tr>
<tr>
<td>1-6 years</td>
<td>400</td>
</tr>
<tr>
<td>7 years and above</td>
<td>600</td>
</tr>
</tbody>
</table>

(Note: 1 μg = 1/1000 mg)

b) 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. You have already learnt that vitamin A is fat-soluble and can be stored for longer period in the liver. The women during pregnancy 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. 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 children. These are known to decrease the absorption of vitamin A and lead to 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 vitamin A injections into the muscle (intramuscularly) in the dose of 100,000 IU*. On the next day 200,000 IU of vitamin A is given by mouth.

(* About 3 International Units (IU) of Vitamin A is equivalent to 1 microgram (µg) of Vitamin A.)

Children with night blindness, conjunctival xerosis and Bitot spots are treated with a massive oral (by mouth) dose of 200,000 IU of vitamin A.

How to prevent Vitamin A deficiency?

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 make sure that communities 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 which, as you know is the 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. However, improvement of Indian diets requires extensive and continuous nutrition education programmes.

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 liver for prolonged periods and will be available to the body. 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 of 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 (200,000 IU) 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 bring down the 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 23 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?
   - ...
   - ...
   - ...

17.4 LET US SUM 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 muscular 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 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 particularly 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, carrot, yellow pumpkin, papaya and mango. Blindness due to xerophthalmia can be prevented by administration of massive dose of vitamin A (i.e. 200,000 IU of Vitamin A) every six months to the children between 1 and 5 years of age.

17.5 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 (µg) : 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.

17.6 ANSWERS TO CHECK YOUR PROGRESS EXERCISES

Check Your Progress Exercise 1

1) Features

<table>
<thead>
<tr>
<th></th>
<th>Kwashiorkor</th>
<th>Marasmus</th>
</tr>
</thead>
<tbody>
<tr>
<td>Oedema</td>
<td>Present</td>
<td>Absent</td>
</tr>
<tr>
<td>Muscle wasting</td>
<td>Moderate</td>
<td>Severe</td>
</tr>
<tr>
<td>Hair changes</td>
<td>Present</td>
<td>Absent</td>
</tr>
<tr>
<td>Body weight</td>
<td>60% of normal</td>
<td>50% of normal</td>
</tr>
<tr>
<td>Moon face</td>
<td>Present</td>
<td>Absent</td>
</tr>
</tbody>
</table>

2) a) Severe (i.e. \(4.7 \times 100 = 49.5\%\) which is less than 50% of the normal weight 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 by pregnant women to increase birth weight of their offspring.
   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 and poor hygiene, Ignorance, Wrong child 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

4) Give daily one of the inexpensive local foods like green leafy vegetables, carrots, yellow pumpkin, papaya or mango.
   • Provide massive oral dose of Vitamin A (200,000 IU) once every six months.