Vitamin A

Learning Objectives

At the end of lecture the student should able to:

- Identify the importance of vitamin A
- Discuss the risk factors and important clinical features of Vitamin A deficiency.
- Outline the management steps of vitamin A deficiency
- Define hypervitaminosis A and distinguish its clinical manifestation
- To state the causes of hemorrhagic disease of newborn
- Classify types of hemorrhagic disease of newborn
- Identify how to prevent hemorrhagic disease of newborn

Overview about vitamin A

Vitamin A is a fat-Soluble vitamin.
Most of total body vitamin A Store in the liver. Stored vitamin A is released from the liver into the circulation as retinol bound to its specific transport protein, retinol-binding protein (RBP).

Function of Vitamin A and mechanisms of action

It regulates many genes that are involved in the fundamental biologic activities of cells, such as cell division, cell death, and cell differentiation.
The term *retinoids* includes both natural and synthetic compounds with vitamin A activity.
Retinoic acid affects many physiologic processes, including reproduction, growth, embryonic and fetal development, and bone development, in addition to respiratory, gastrointestinal, hematopoietic, and immune functions. Vitamin A play important role in immune function and host defense .
The mechanism of vitamin A action in vision is that it play a role in synthesis of Rhodopsin and Iodopsin .
Vitamin A is important to maintenance of epithelial functions. In the intestines a normal goblet cell function is an effective barrier against pathogens that can cause diarrhea. Similarly in the respiratory tract, a mucus-secreting epithelium is essential for the disposal of inhaled pathogens and toxicants.

- **Retinol (Vitamin A) 1µg = 3,3 IU vitamin A**

Vitamin A and measles vaccine

Vitamin A enhanced the antibody response to measles vaccine given at 9 months of age. The children who had received vitamin A with their measles vaccine were more protected against measles at 6-8 years of age.

Vitamin A treatment of children with measles

Associated with reductions in morbidity and mortality (morbidity especially from pneumonia).

Vitamin A deficiency

Patient at risk for Vitamin A deficiency

- Common in many developing countries and are often associated with global malnutrition .
- Malnutrition, because of the impaired synthesis of retinol-binding protein
- As a complications in children with various chronic disorders or diseases.

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Note: Vitamin A deficiency is the commonest cause of blindness in developing Countries. It causes eye damage (xerophthalmia), which may progress from night blindness to corneal ulceration and scarring.

Sources of vitamin A

- Diet is the main source of vitamin A
- Liver, fish liver oils
- Dairy products
- Vegetables
- Egg yolk
- fortified margarine,

Dietary reference intakes for infants andchildren.

Table 48-2 Dietary Reference Intakes for Vitamin A in Children

AGE RANGE	RECOMMENDED DIETARY ALLOWANCE (RDA) (µg retinol equivalents per day)	UPPER LEVEL (UL) (µg retinol equivalents per day)	COMMENTS
0-6 mo	400	600	The recommended intake for infants is an adequate intake, based on the amount of vitamin A normally present in breast milk
7-12 mo	500	600	
1-3 yr	300	600	The UL applies only to preformed vitamin A (retinol).
4-8 yr	400	900	
9-13 yr	600	1,700	
14-18 yr	900 male; 700 female	2,800	

Clinical features of vitamin A deficiency

- Dry, scaly, hyperkeratotic patches, commonly on the arms, legs, shoulders, and buttocks.
- Poor growth
- eye lesions : An early symptom is delayed adaptation to the dark; later when vitamin A deficiency is more advanced, it leads to night blindness
- Photophobia
- Xerophthalmia
- (keratomalacia
- (Bitot spots (keratinized conjunctiva)
- (conjunctival xerosis)
- Diarrhea, susceptibility to infections, anemia, apathy, mental retardation

Treatment of Vitamin A deficeincy

A daily supplement of 1,500 µg of vitamin A is sufficient for treating latent vitamin A deficiency. Xerophthalmia is treated by giving 1,500 µg/kg body weight orally for 5 days followed by intramuscular injection of 7,500 µg of vitamin A in oil, until recovery

HYPERVITAMINOSIS A

Definition:

Acute hypervitaminosis A, occure after consumption of a single large (30-60 mg dose) of vitamin A. Features may include nausea, vomiting, and drowsiness; less-common symptoms include diplopia, papilledema, cranial nerve palsies, and other symptoms suggesting pseudotumorcerebri.

Chronic hypervitaminosis A results from excessive ingestion of preformed vitamin A (retinol or retinyl ester), generally for several weeks or months. Toxicity can be induced in adults and children with chronic daily intakes of 15,000 μg and 6,000 μg, respectively.

Clinical features of hypervitaminosis A

- Increased intracranial pressure ; pseudotumor cerebri
- Bone abnormalities; swelling of the bones
- Enlargement of the liver and spleen
- Seborrheic cutaneous lesions
- In young children, toxicity is associated with vomiting and bulging fontanels. An affected child has anorexia, pruritus, and a lack of weight gain.

Radiographs in hypervitaminosis A show hyperostosis affecting several long bones

Hemorrhagic Disease of the Newborn

Normally in all newborn there is transient deficiency of vitamin K–dependent factors, (II, VII, IX, and X) probably because lack of free vitamin K from the mother and absence of the bacterial intestinal flora normally responsible for the synthesis of vitamin K.

	EARLY-ONSET DISEASE	CLASSIC DISEASE	LATE-ONSET DISEASE
Age	0-24 hr	2-7 days	1-6 mo
Bleeding site	Cephalohematoma Intracranial Gastrointestinal Umbilicus Circumcision Injection sites , mucocutaneos		
Etiology/risks	Maternal drugs (phenobarbital, phenytoin, warfarin, rifampin,) that interfere with vitamin K	Vitamin K deficiency Breastfeeding	Cholestasis , malabsorption of vitamin K (biliary atresia, cystic fibrosis, hepatitis) Warfarin ingestion
Prevention	• Intramuscular administration of 1 mg of vitamin K at the time of birth • Oral vitamin K (birth, discharge, 3-4 wk: 1-2 mg)		
Treatment	• Slow intravenous infusion of 1-5 mg of vitamin K1, with improvement in coagulation defects and cessation of bleeding noted within a few hours. • If no response, transfusion of fresh-frozen plasma or whole blood may be needed.		