

HIGH-YIELD NUTRITION NOTES

FOR USMLE STEP 1

By

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Complete Vitamin Deficiencies & Toxicities with case
scenario examples

Calorie counts

Refeeding and Starvation

Protein energy malnutrition

FAT-SOLUBLE VITAMINS

VITAMIN A (RETINOL)

DEFICIENCY

Pathophysiology

- Rhodopsin production fails → rods can't detect light in dim conditions → night blindness (first sign)
- Epithelial cells lose mucus-secreting ability → squamous metaplasia → dry eyes, respiratory infections
- Conjunctival/corneal drying → xerophthalmia → keratomalacia → blindness

Risk Factors

- Chronic fat malabsorption (Crohn's, CF, celiac, chronic pancreatitis)
- Severe protein-energy malnutrition
- Measles infection (depletes vitamin A)
- Developing countries (leading cause of preventable blindness in children)

Clinical Presentation

Early Signs:

- Night blindness (nyctalopia) - FIRST symptom
 - Difficulty seeing in dim light
 - Trouble with dark adaptation

Progressive Eye Disease (Xerophthalmia Spectrum):

1. Conjunctival xerosis - dry, rough conjunctiva
2. Bitot spots - foamy, triangular white patches on bulbar conjunctiva (pathognomonic!)
3. Corneal xerosis - dry, hazy cornea
4. Corneal ulceration/keratomalacia - corneal melting → IRREVERSIBLE BLINDNESS

Other Features:

- Dry, scaly skin (follicular hyperkeratosis)
- Increased infections (especially respiratory, measles)
- Poor wound healing
- Growth retardation in children

Diagnosis

- Serum retinol < 20 µg/dL (normal: 30-70 µg/dL)
- Clinical diagnosis in endemic areas (Bitot spots are pathognomonic)
- Response to treatment - night blindness improves within days

Treatment

Oral vitamin A:

- Adults: 200,000 IU PO once, repeat next day and at 4 weeks
- Children: 100,000-200,000 IU (age-dependent)
- With corneal involvement: Give immediately to prevent blindness
- Prevention: Vitamin A supplementation in at-risk populations (measles, malnutrition)

USMLE Case Scenario

"A 4-year-old boy from rural Nepal is brought to clinic by his mother who reports he has difficulty seeing at dusk and frequently bumps into objects when indoors. On examination, you notice bilateral foamy, triangular white patches on the temporal conjunctiva. His diet consists mainly of rice and lentils with minimal vegetables or animal products. What is the most likely diagnosis?"

Answer: Vitamin A deficiency (xerophthalmia with Bitot spots)

Key teaching points:

- Night blindness = FIRST symptom
- Bitot spots = PATHOGNOMONIC
- High-risk: poor diet, no animal products/yellow vegetables
- Treat urgently to prevent irreversible blindness

TOXICITY

Pathophysiology

- Fat-soluble → accumulates in liver
- Acute: single massive dose (>300,000 IU)
- Chronic: prolonged excessive intake (>10,000 IU/day for months)

Causes

- Excessive supplementation (most common)
- Isotretinoin (Accutane) for acne
- Polar bear liver consumption (classic boards question!)
- Over-supplementation during pregnancy → teratogenicity

Clinical Presentation

Acute Toxicity:

- Nausea, vomiting, headache
- Increased intracranial pressure → pseudotumor cerebri (benign intracranial hypertension)
- Blurred vision, papilledema
- Dizziness, irritability

Chronic Toxicity:

- Dry, rough skin with desquamation
- Alopecia (hair loss)
- Bone/joint pain
- Hepatomegaly, cirrhosis (with very prolonged use)
- Hypercalcemia
- Teratogenicity: craniofacial, cardiac, thymic, CNS defects

Diagnosis

- Serum retinol > 100 µg/dL
- Elevated serum calcium (sometimes)

- Imaging: If pseudotumor cerebri → MRI/MRV to rule out venous thrombosis; LP shows ↑ opening pressure with normal CSF

Treatment

- STOP vitamin A supplementation
- Supportive care
- Pseudotumor cerebri: acetazolamide, weight loss, serial LPs
- Pregnancy: Discontinue immediately; fetal effects irreversible

USMLE Case Scenario

"A 28-year-old woman presents with severe headaches, nausea, and blurred vision for 2 weeks. She has been taking high-dose vitamin A supplements (50,000 IU daily) for 'skin health' for the past 3 months. Examination reveals bilateral papilledema and dry, peeling skin. Neuroimaging shows no mass lesions. Lumbar puncture reveals an opening pressure of 350 mm H₂O with normal CSF analysis. What is the most likely diagnosis?"

Answer: Vitamin A toxicity causing pseudotumor cerebri

Key teaching points:

- Fat-soluble vitamins accumulate → toxicity
- Pseudotumor cerebri: ↑ICP without structural cause
- Women of childbearing age: PREGNANCY TEST! (teratogenic)
- Treatment: STOP vitamin A

Boards Buzzwords

- "Foamy triangular white patches on conjunctiva" = Bitot spots = Vitamin A deficiency
- "Polar bear liver" = Vitamin A toxicity
- "Night blindness + malnourished child" = Vitamin A deficiency
- "Isotretinoin + pregnancy" = Teratogenicity (vitamin A analog)
- "Pseudotumor cerebri + supplements" = Vitamin A toxicity

VITAMIN D (CALCIFEROL)

DEFICIENCY

Pathophysiology

- Vitamin D → 25-OH vitamin D (liver) → 1,25-(OH)₂ vitamin D (kidney) = active form
- Active vitamin D increases calcium absorption from gut and bone resorption
- Deficiency → ↓ calcium absorption → ↓ serum calcium → ↑ PTH (secondary hyperparathyroidism)
- ↑ PTH → mobilizes calcium from bones → inadequate bone mineralization
- Children: Defective mineralization of growing bones = RICKETS
- Adults: Defective mineralization of mature bones = OSTEOMALACIA

Risk Factors

- Limited sun exposure (homebound elderly, veiled individuals, winter, high latitudes)
- Dark skin (melanin blocks UV penetration)
- Malabsorption (celiac, Crohn's, chronic pancreatitis, gastric bypass)
- Chronic kidney disease (↓ 1α-hydroxylase)
- Anticonvulsants (phenytoin, phenobarbital - induce CYP450)
- Exclusively breastfed infants (breast milk low in vitamin D)
- Strict vegan diet without fortified foods

RICKETS (Children)

Clinical Presentation

Skeletal Deformities:

- Craniotabes - soft skull bones (ping-pong ball sensation)
- Frontal bossing - prominent forehead
- Delayed fontanelle closure
- Rachitic rosary - beading at costochondral junctions (looks like beads on chest)
- Harrison groove - horizontal indentation along lower chest
- Bowed legs (genu varum) or knock-knees (genu valgum)
- Wrist/ankle widening - metaphyseal expansion

- Delayed motor milestones (sitting, walking)
- Short stature

Other Features:

- Muscle weakness, hypotonia ("floppy baby")
- Bone pain
- Dental defects
- Increased fracture risk
- Tetany/seizures (if severe hypocalcemia)

Diagnosis

- ↓ 25-OH vitamin D (<20 ng/mL = deficiency; <12 ng/mL = severe)
- ↓ Calcium (or low-normal)
- ↓ Phosphate
- ↑ PTH (secondary hyperparathyroidism)
- ↑ Alkaline phosphatase (↑↑↑ - bone turnover marker)

X-ray findings:

- Widened, frayed, cupped metaphyses
- Loss of sharp metaphyseal border
- Decreased bone density
- Looser zones (pseudofractures) - rare in children

Treatment

Vitamin D supplementation:

- Ergocalciferol (D2) or cholecalciferol (D3): 2,000-6,000 IU daily for 6-12 weeks
- Calcium supplementation: 500-1,000 mg/day
- Monitor: 25-OH vitamin D levels, calcium, phosphate, PTH, alkaline phosphatase
- Prevention: 400 IU/day for infants, 600 IU/day for children

VITAMIN K (PHYLLOQUINONE)

DEFICIENCY

Pathophysiology

- Vitamin K is essential cofactor for γ -carboxylation of clotting factors II, VII, IX, X (remember: "1972") and proteins C & S
- Deficiency → undercarboxylated, non-functional clotting factors → COAGULOPATHY
- Factor VII has shortest half-life (6 hours) → PT/INR rises FIRST
- Also needed for bone proteins (osteocalcin) and anticoagulant proteins (proteins C & S)

Risk Factors

Newborns (most susceptible):

- Low placental transfer, sterile gut (no vitamin K-producing bacteria yet), low breast milk vitamin K

Fat malabsorption:

- Biliary obstruction, cholestatic liver disease, cystic fibrosis, celiac, Crohn's, chronic pancreatitis
- Antibiotic use (broad-spectrum) - kills gut bacteria that produce vitamin K
- Warfarin (vitamin K antagonist)
- Malnutrition, TPN without vitamin K supplementation

HEMORRHAGIC DISEASE OF THE NEWBORN

Clinical Presentation

Three Forms:

1. Early (0-24 hours):

- Maternal drug use (warfarin, anticonvulsants, rifampin)
- Severe bleeding at birth

2. Classic (2-7 days) - MOST COMMON:

- Breastfed infants without prophylaxis
- GI bleeding (melena, hematemesis)