



Vitamin a Deficiency: An Updated Review for Healthcare Professionals and Nutrition Practitioners

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Abstract:

Background: Vitamin A deficiency (VAD) is a significant global health issue, particularly affecting children and pregnant women in low-income regions. Vitamin A is essential for vision, immune function, and cellular development, and its deficiency can lead to severe complications, including blindness, increased susceptibility to infections, and high mortality rates. While VAD is rare in developed countries, it remains prevalent in resource-poor settings due to inadequate dietary intake and recurrent infections. Public health interventions, such as supplementation, food fortification, and biofortification, have been implemented to address this issue.

Aim: This review aims to provide healthcare professionals and nutrition practitioners with an updated understanding of VAD, including its etiology, epidemiology, pathophysiology, clinical presentation, diagnostic evaluation, treatment, and prevention strategies.

Methods: The review synthesizes current literature and guidelines on VAD, focusing on its global prevalence, risk factors, clinical manifestations, and management. It highlights the roles of dietary assessment, supplementation, and public health interventions in addressing VAD.

Results: VAD disproportionately affects children under five and pregnant women in developing countries, contributing to significant morbidity and mortality. Early symptoms include night blindness and xerophthalmia, which can progress to irreversible blindness if untreated. Serum retinol levels are commonly used for diagnosis, though liver biopsy remains the gold standard. Treatment involves vitamin A

supplementation (VAS), with dosing tailored to age, severity, and regional guidelines. Prevention strategies include dietary diversification, food fortification, and biofortification of staple crops.

Conclusion: VAD is a preventable condition with severe consequences if left untreated. Effective management requires a multifaceted approach, including supplementation, dietary education, and public health initiatives. Collaboration among healthcare professionals, nutrition practitioners, and policymakers is essential to reduce the global burden of VAD.

Keywords: Vitamin A deficiency, xerophthalmia, supplementation, food fortification, biofortification, public health.

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Introduction:

Vitamin A is a critical fat-soluble vitamin that plays a vital role in numerous physiological processes, including cell development, metabolism, immune function, vision, and reproductive health [1][2]. Its deficiency, known as vitamin A deficiency (VAD), is a significant global health issue, particularly affecting young children in low-income regions. VAD is associated with high morbidity and mortality rates due to its impact on essential bodily functions. Natural sources of vitamin A include dark leafy greens, orange-colored vegetables, dairy products, liver, and fish [3]. The absorption of vitamin A occurs in the duodenum, where it is hydrolyzed by pancreatic and intestinal enzymes and emulsified with dietary fats and bile acids [4]. Once absorbed, the majority of vitamin A is stored in hepatic stellate cells, with additional reserves in adipose tissue and the pancreas [5][6].

The recommended dietary allowance (RDA) for vitamin A, as established by the Institute of Medicine, varies by age, sex, and physiological status. For healthy adults, the RDA is 700 micrograms per day for women and 900 micrograms per day for men [7][8]. Children, pregnant women, and lactating women have higher RDAs, ranging from 300 to 900, 770, and 1300 micrograms per day, respectively. To prevent symptomatic VAD in children aged 1 to 5 years, a minimum intake of approximately 200 micrograms per day is required. Serum retinol levels are a reliable indicator of vitamin A status, with deficiency defined as a concentration of fewer than 20 micrograms per deciliter ($\mu\text{g}/\text{dL}$) [8]. Ocular symptoms of VAD, such as night blindness and xerophthalmia, typically manifest at serum retinol levels below 10 $\mu\text{g}/\text{dL}$ [8]. Vitamin A is obtained from the diet in two primary forms: carotenoids from plant sources and retinoids from animal products. Carotenoids, such as beta-carotene, are provitamin A compounds that the body can convert into active vitamin A. Retinoids, including retinol and retinyl esters, are preformed active vitamin A. The absorption efficiency of retinoids is high, ranging from 75% to 100%, whereas carotenoid absorption varies significantly depending on the food matrix and the specific type of carotenoid [9][10]. The Centers for Disease Control and Prevention (CDC) estimates a 12:1 ratio of beta-carotene to retinol absorption in a mixed diet of fruits and vegetables [7]. This conversion ratio highlights the challenge of obtaining sufficient vitamin A from plant-based sources alone, particularly in populations with limited access to animal products or carotenoid-rich vegetables.

In many developing countries, diets are often deficient in vitamin A due to limited consumption of meat, dairy, and carotenoid-rich vegetables. This dietary inadequacy exacerbates the prevalence of VAD, particularly among vulnerable groups such as children and pregnant women. Public health interventions, including dietary diversification, food fortification, and supplementation programs, have been implemented to address VAD in these regions. For example, high-dose vitamin A supplementation is a widely adopted strategy to reduce deficiency-related morbidity and mortality in children. However, sustainable long-term solutions require improving access to diverse and nutrient-rich diets, alongside education on the importance of vitamin A for health. In conclusion, vitamin A is indispensable for maintaining essential physiological functions, and its deficiency poses a significant public health challenge, particularly in resource-limited settings. Addressing VAD requires a multifaceted approach, combining immediate interventions like supplementation with long-term strategies to improve dietary quality and

accessibility. Ensuring adequate vitamin A intake is crucial for reducing the global burden of VAD and its associated health consequences.

Etiology

In resource-poor regions, the primary etiology of vitamin A deficiency (VAD) is insufficient nutrition, often exacerbated by chronic inflammation from recurrent gastrointestinal (GI) infections. These infections impair nutrient absorption and utilization, leading to widespread micronutrient deficiencies, including VAD [11]. Children in these regions are particularly vulnerable due to the frequent co-occurrence of zinc deficiency, which is essential for vitamin A absorption and the synthesis of retinol-binding protein (RBP), the transport protein for vitamin A in the bloodstream [12]. Additionally, measles, endemic in many of these areas, causes a rapid decline in serum retinol levels by more than 30%. This occurs because measles infection reduces RBP synthesis and increases urinary excretion of vitamin A, while the damage to GI epithelial tissues further elevates physiological demand for the vitamin [13].

Maternal nutrition significantly influences vitamin A levels in breast milk. In resource-poor settings, breast milk often contains only enough vitamin A to meet an infant's minimum daily requirements, leaving no surplus for building liver reserves. This results in a high prevalence of VAD shortly after weaning [14]. In contrast, VAD is exceedingly rare in developed countries due to the widespread availability of vitamin A-rich foods, better sanitation, access to clean water, and advanced healthcare systems. Cases of VAD in developed nations are typically linked to primary or secondary intestinal malabsorptive disorders rather than dietary insufficiency. In the developed world, VAD is most commonly associated with pancreatic, liver, and intestinal pathologies. Chronic inflammatory conditions, such as inflammatory bowel disease (IBD), mirror the effects of frequent GI infections seen in developing regions. IBD causes persistent inflammation of the intestinal mucosa, which, combined with reduced oral intake, can lead to VAD [15]. Chronic liver disease is another significant contributor to VAD, although the exact mechanisms remain unclear. Proposed explanations include decreased bile acid production, which is necessary for vitamin A absorption, and altered storage patterns in the liver [6].

Pancreatic insufficiency, often seen in conditions like cystic fibrosis or chronic pancreatitis, can also lead to VAD due to inadequate production of digestive enzymes, including hydrolases required for vitamin A absorption [16]. Bariatric surgeries, particularly those that bypass the duodenum, are another cause of VAD in developed countries. These procedures are designed to limit fat absorption but inadvertently reduce the uptake of fat-soluble vitamins, including vitamin A. Premature infants are another at-risk group due to their immature GI tracts, limited vitamin A stores, and heightened nutritional needs during rapid growth and development [17]. In summary, the etiology of VAD varies significantly between resource-poor and developed regions. In low-income settings, poor nutrition, recurrent infections, and coexisting micronutrient deficiencies are the primary drivers, while in developed countries, VAD is more often linked to malabsorptive disorders, chronic diseases, and surgical interventions. Understanding these distinct etiological factors is crucial for designing targeted interventions to address VAD in different populations.

Epidemiology

Vitamin A deficiency (VAD) remains a significant global health concern, particularly in developing countries, where it disproportionately affects children under five years of age. Globally, an estimated 30% of children in this age group suffer from VAD, contributing to approximately 2% of all deaths among young children [18]. Although the prevalence of VAD has been decreasing due to public health interventions, it remains a leading cause of preventable blindness and increased susceptibility to infections in low-income regions. Pregnant and lactating women are also at heightened risk of VAD due to their increased physiological demands. For instance, a 2019 study in rural Ethiopia found that 76% of lactating mothers were vitamin A deficient [19]. VAD does not exhibit a gender predilection, affecting males and females equally [20][21]. In contrast, VAD is rare in the general population of the United States (US), with an estimated prevalence of only 0.3% as of 2013. In fact, vitamin A toxicity is more common in the US than deficiency, reflecting the widespread availability of vitamin A-rich foods and fortified products [7]. Symptomatic VAD in the US is typically associated with malabsorptive disorders or severely restrictive diets. For example, 16% of children

with inflammatory bowel disease (IBD) in the US are vitamin A deficient at diagnosis, with a higher prevalence observed in Crohn disease compared to ulcerative colitis [15].

Patients with chronic liver disease, particularly those with cirrhosis severe enough to require transplantation, exhibit a 70% prevalence of VAD. The severity of cirrhosis correlates positively with the likelihood of VAD, likely due to impaired storage and metabolism of vitamin A [22]. Similarly, chronic exocrine pancreatic insufficiency is associated with VAD, affecting 35% of patients despite 84% of them receiving pancreatic enzyme replacement therapy [16]. Bariatric surgery, especially procedures like biliopancreatic diversion that limit fat absorption, also significantly increases the risk of VAD. Studies show that 70% of patients develop VAD within three years post-procedure [23]. Premature infants are another high-risk group for VAD due to their underdeveloped gastrointestinal tracts, limited vitamin A stores, and increased nutritional needs during rapid growth. At birth, 66% of premature infants are vitamin A deficient, and by 36 weeks post-menstrual age, this figure rises to 82% [17]. In summary, the epidemiology of VAD varies widely between developing and developed countries. In low-income regions, VAD is predominantly a public health issue affecting young children and lactating women, while in developed nations, it is largely associated with malabsorptive conditions, chronic diseases, and surgical interventions. Understanding these epidemiological patterns is essential for implementing targeted prevention and treatment strategies to address VAD in diverse populations.

Pathophysiology

Vitamin A plays a critical role in several physiological processes, including the regeneration of visual pigments, maintenance of mucosal membranes, and regulation of immune function. Its deficiency disrupts these processes, leading to a cascade of clinical manifestations. One of the earliest and most recognizable symptoms of vitamin A deficiency (VAD) is night blindness, which occurs due to impaired regeneration of rhodopsin, the visual pigment in retinal rods. Rhodopsin is essential for vision in low-light conditions, and its insufficient regeneration results in diminished night vision [24][25]. If VAD persists, the retinal rods degenerate, leading to xerophthalmia, a condition characterized by dryness and thickening of the conjunctiva and cornea. Without intervention, xerophthalmia can progress to corneal ulceration, scarring, and irreversible blindness [26].

Beyond its role in vision, vitamin A is vital for maintaining the integrity of epithelial tissues, including the mucosal membranes of the gastrointestinal (GI) and respiratory tracts. In VAD, these membranes become dry (xerosis) and prone to breakdown, compromising their barrier function. This mucosal damage increases susceptibility to infections, particularly in the GI and respiratory systems [27][28]. The immune system is also adversely affected, as vitamin A is essential for the normal functioning of both innate and adaptive immunity. Deficiency impairs the production and function of immune cells, such as lymphocytes and macrophages, and reduces the body's ability to combat infections [29]. Additionally, VAD contributes to anemia of chronic inflammation. The deficiency exacerbates inflammatory responses and disrupts iron metabolism, leading to reduced erythropoiesis and the development of anemia. This type of anemia is characterized by functional iron deficiency, where iron is sequestered in storage sites rather than being available for red blood cell production [30]. In summary, the pathophysiology of VAD involves a multifaceted disruption of visual, epithelial, and immune functions. Night blindness and xerophthalmia result from impaired visual pigment regeneration and retinal degeneration, while mucosal membrane breakdown and immune dysfunction increase the risk of infections. Furthermore, VAD contributes to anemia of chronic inflammation by altering iron metabolism and exacerbating inflammatory responses. Understanding these mechanisms highlights the importance of addressing VAD to prevent its wide-ranging and severe health consequences.

History and Physical

A thorough history and physical examination are crucial for identifying vitamin A deficiency (VAD). Clinicians should maintain a high index of suspicion for VAD in patients with risk factors such as malabsorptive conditions (e.g., inflammatory bowel disease [IBD], chronic gastrointestinal [GI] infections, pancreatic insufficiency, or cirrhosis), prematurity, a history of measles infection, residence in resource-

poor regions, or pregnancy and lactation in the context of poor nutrition. These factors significantly increase the likelihood of VAD due to impaired absorption, increased physiological demand, or inadequate dietary intake. The clinical presentation of chronic VAD typically follows a progressive course. Early symptoms often include night blindness (nyctalopia), which results from impaired regeneration of rhodopsin in retinal rods. Patients may also experience recurrent infections of the GI, pulmonary, and urinary tracts due to compromised mucosal integrity and immune dysfunction. Dermatological manifestations, such as xeroderma (dry, scaly skin) and phrynoderma (follicular hyperkeratosis), may develop, particularly on extensor surfaces, shoulders, and buttocks [30][31].

As VAD progresses, ocular signs become more pronounced. Xerophthalmia, a hallmark of advanced VAD, manifests with Bitot spots—foamy, triangular, or oval lesions on the conjunctiva—and conjunctival xerosis, characterized by a dry, wrinkled appearance of the conjunctiva. Without intervention, the condition worsens to corneal xerosis (dryness and clouding of the cornea), corneal ulceration, and ultimately keratomalacia, where the cornea softens and perforates, leading to irreversible blindness [26]. In cases of acute VAD, particularly during measles infection, patients may present with rapid-onset corneal xerosis and ulceration without the preceding symptoms of night blindness or Bitot spots. Phrynoderma, though classically associated with VAD, is not pathognomonic and can occur in the context of other nutritional deficiencies, such as essential fatty acid or vitamin B complex deficiencies. Therefore, its presence should prompt a broader nutritional assessment [31]. In summary, a detailed history and physical examination are essential for diagnosing VAD. Key findings include night blindness, recurrent infections, xeroderma, phrynoderma, and ocular signs such as Bitot spots, conjunctival xerosis, and corneal changes. Recognizing these clinical features, particularly in high-risk populations, enables timely intervention to prevent the severe complications of VAD, including blindness and systemic morbidity.

Evaluation

The evaluation of vitamin A deficiency (VAD) relies on a combination of clinical findings and laboratory testing. In cases where classical signs of VAD are present, such as xerophthalmia, a clinical diagnosis can often be made without additional testing, as xerophthalmia is nearly pathognomonic for VAD [32]. However, in patients with less clear presentations or early-stage deficiency, laboratory testing is essential for confirmation. The most commonly used laboratory test is serum retinol concentration, with deficiency defined as levels below 20 micrograms per deciliter ($\mu\text{g}/\text{dL}$) [8]. While this test is widely accessible, it has limitations. Serum retinol levels may remain within the normal range even when total body stores are low, as hepatic reserves can maintain circulating retinol levels until advanced deficiency occurs. This makes serum retinol a less sensitive marker for early or mild VAD. The gold standard for assessing total body vitamin A stores is the measurement of liver retinol concentration via liver biopsy. This method provides a direct quantification of hepatic vitamin A reserves, offering the most accurate assessment of nutritional status [33]. However, due to the invasive nature, associated risks, and high cost of liver biopsies, this approach is rarely used in clinical practice and is typically reserved for research settings. In resource-limited settings, where laboratory testing may not be readily available, clinical diagnosis based on history and physical examination remains the primary method for identifying VAD. Public health programs often rely on population-level indicators, such as the prevalence of night blindness or xerophthalmia, to assess the burden of VAD and guide intervention strategies. In summary, the evaluation of VAD involves a combination of clinical assessment and laboratory testing. While xerophthalmia is a highly specific indicator, serum retinol testing is commonly used to confirm the diagnosis, despite its limitations. Liver biopsy, though the most accurate method, is impractical for routine clinical use. Clinicians must consider the context and available resources when evaluating patients for VAD, particularly in high-risk populations.

Treatment / Management

The cornerstone of treatment for vitamin A deficiency (VAD) is vitamin A supplementation (VAS), which has been shown to significantly reduce childhood morbidity and mortality in populations with VAD [34]. VAS is particularly effective in patients with serum retinol concentrations below 20 micrograms per

deciliter ($\mu\text{g}/\text{dL}$). Patients with levels above 30 $\mu\text{g}/\text{dL}$ do not benefit from supplementation and should adhere to the recommended dietary allowance (RDA) for vitamin A [35].

Universal Supplementation in High-Prevalence Regions

In regions with a high prevalence of VAD, the World Health Organization (WHO) recommends universal VAS for specific populations. Key recommendations include:

- **Children 6 to 11 months of age:** A one-time dose of 100,000 international units (IU) of vitamin A.
- **Children 1 to 5 years of age:** Doses of 200,000 IU every 4 to 6 months [36].
- **At-risk pregnant women:** Lower doses are recommended due to concerns about fetotoxicity. The WHO suggests 10,000 IU daily or 25,000 IU weekly for 12 weeks [37].

The WHO no longer recommends universal VAS for children under 6 months of age or postpartum women, as evidence supporting its benefits in these groups is insufficient [38][39][40].

Treatment of Symptomatic VAD

For symptomatic VAD, particularly **xerophthalmia**, the WHO recommends the following VAS dosing:

- **Children under 6 months:** 50,000 IU daily for 2 days, followed by an additional dose after 2 weeks.
- **Children 6 to 12 months:** 100,000 IU daily for 2 days, followed by an additional dose after 2 weeks.
- **Children over 12 months:** 200,000 IU daily for 2 days, followed by an additional dose after 2 weeks [41].

In cases of **severe measles**, the same dosing regimen is recommended, regardless of the patient's known vitamin A status, as measles can rapidly deplete vitamin A stores and exacerbate deficiency [41].

Special Considerations

- **Zinc Deficiency:** Patients with concurrent zinc deficiency may have a poor response to VAS. Therefore, zinc supplementation should be administered alongside VAS to ensure optimal outcomes [12].
- **Malabsorption:** For patients with malabsorptive conditions, such as inflammatory bowel disease (IBD) or pancreatic insufficiency, intramuscular VAS formulations may be considered to bypass the impaired gastrointestinal absorption.

Resource-Rich Settings

In resource-rich countries, VAS strategies are tailored to specific populations:

- **Post-Bariatric Surgery Patients:** These patients are at high risk for VAD due to fat malabsorption. The recommended dose is 10,000 IU daily, with adjustments based on regular monitoring of serum retinol levels. Some patients may require up to 100,000 IU daily to correct deficiency [42].
- **Premature Infants:** While formal guidelines are lacking, studies have shown promising results with VAS in this population:
 - **Very low birth weight neonates:** Supplementation with 10,000 IU every other day for 4 weeks reduced all-cause mortality by 56% and decreased rates of oxygen requirement, sepsis, patent ductus arteriosus (PDA), and hospital stay [43].
 - **Extremely premature infants:** Supplementation with 1,500 IU daily significantly reduced the incidence of retinopathy of prematurity (1.6% vs. 6.9%) and bronchopulmonary dysplasia by nearly 50% [44].

Case-by-Case Management

For VAD associated with other malabsorptive processes, such as chronic liver disease or pancreatic insufficiency, treatment should be individualized based on the underlying condition and severity of

deficiency. VAS is a highly effective intervention for treating and preventing VAD, particularly in high-prevalence regions. In resource-rich settings, tailored dosing strategies are necessary for at-risk populations, such as post-bariatric surgery patients and premature infants. Clinicians must consider the patient's clinical context, underlying conditions, and nutritional status when determining the appropriate VAS regimen. Regular monitoring and adjunctive treatments, such as zinc supplementation, may be required to optimize outcomes.

Differential Diagnosis

When evaluating a patient for suspected vitamin A deficiency (VAD), it is crucial to consider other conditions that may present similar signs and symptoms. A thorough differential diagnosis ensures accurate identification and appropriate management of the underlying cause. Key conditions to consider include:

1. Night Blindness (Nyctalopia)

- **Retinitis Pigmentosa:** A genetic disorder characterized by progressive degeneration of retinal photoreceptors, leading to night blindness, peripheral vision loss, and eventual central vision impairment [45].
- **Retinal Dystrophies:** Rare inherited conditions affecting the retina, such as congenital stationary night blindness, which can present with night blindness without systemic symptoms [46].
- **Cataracts:** Opacification of the lens that can impair night vision due to reduced light transmission.
- **Myopia:** Severe nearsightedness may cause difficulty with night vision, though it is typically accompanied by other refractive errors.

2. Ocular Manifestations

- **Niacin Deficiency (Pellagra):** While Bitot spots are classically associated with VAD, they can also occur in niacin deficiency, which is characterized by dermatitis, diarrhea, and dementia [47].
- **Pinguecula:** A benign, yellowish growth on the conjunctiva that may resemble Bitot spots but is not associated with nutritional deficiencies.
- **Pterygium:** A fleshy growth of conjunctival tissue that can extend onto the cornea, sometimes mimicking Bitot spots in appearance.

3. Dermatological Manifestations

- **Essential Fatty Acid Deficiency:** Can cause dry, scaly skin (xeroderma) and follicular hyperkeratosis (phrynoderma), similar to VAD.
- **Zinc Deficiency:** May be present with skin lesions, impaired wound healing, and immune dysfunction, overlapping with some symptoms of VAD.
- **Vitamin B Complex Deficiency:** Can lead to skin changes, such as seborrheic dermatitis or hyperkeratosis, which may be confused with VAD-related dermatological findings.

4. Immune Dysfunction and Recurrent Infections

- **Primary Immunodeficiency Disorders:** Conditions such as common variable immunodeficiency (CVID) or chronic granulomatous disease (CGD) can cause recurrent infections, mimicking the immune dysfunction seen in VAD.
- **HIV/AIDS:** Chronic immune suppression can lead to frequent infections and nutritional deficiencies, including VAD.

5. Anemia of Chronic Inflammation

- **Chronic Diseases:** Conditions such as chronic kidney disease, rheumatoid arthritis, or malignancies can cause anemia of chronic inflammation, which may overlap with VAD-associated anemia.
- **Iron Deficiency Anemia:** Often presents with fatigue and pallor, but without the ocular or dermatological signs of VAD.

6. Malabsorptive Disorders

- **Celiac Disease:** Can lead to multiple nutrient deficiencies, including VAD, due to impaired absorption in the small intestine.
- **Chronic Pancreatitis:** May cause fat-soluble vitamin deficiencies, including VAD, due to insufficient pancreatic enzyme production.

The differential diagnosis for VAD includes a wide range of conditions that can present with overlapping signs and symptoms, such as night blindness, ocular lesions, skin changes, immune dysfunction, and anemia. Clinicians must carefully evaluate the patient's history, physical examination findings, and laboratory results to distinguish VAD from other potential causes. Accurate diagnosis is essential for implementing targeted treatment and preventing complications associated with VAD or other underlying conditions.

Prognosis

The prognosis of vitamin A deficiency (VAD) is highly dependent on the severity of the disease at the time of treatment initiation [26]. Early intervention significantly improves outcomes, while delayed treatment can lead to irreversible complications.

Subclinical VAD

Patients with subclinical VAD (low serum retinol levels without overt symptoms) have an excellent prognosis if treated promptly. Supplementation typically leads to rapid improvement, often within a week, with no long-term sequelae [48].

Early Ophthalmologic Manifestations

In cases where VAD presents early ophthalmologic signs, such as night blindness, conjunctival xerosis, or Bitot spots, the prognosis remains favorable with timely treatment. These symptoms are usually resolved completely within 2 months of vitamin A supplementation [49][26]. However, if treatment is delayed, the risk of progression to more severe ocular complications increases.

Advanced Ophthalmologic Manifestations

Patients with corneal xerosis or ulceration are at risk of permanent vision loss due to corneal scarring, even with appropriate treatment. These advanced stages of VAD are associated with significant morbidity and highlight the importance of early diagnosis and intervention [26].

Increased Susceptibility to Infections

The onset of visual manifestations in VAD is often accompanied by an increased susceptibility to infections, particularly gastrointestinal and pulmonary infections, due to compromised mucosal integrity and immune dysfunction. In **preschool children**, the presence of ophthalmologic signs (e.g., night blindness, Bitot spots) is associated with significantly higher mortality rates compared to children with subclinical VAD [26]:

- Children with night blindness have three times the mortality rate of those with subclinical VAD.
- Children with both night blindness and Bitot spots have nine times the mortality rate of those with subclinical VAD.
- Nearly two-thirds of children with keratomalacia (corneal softening and ulceration) die within months, underscoring the life-threatening nature of advanced VAD [26].

Mortality and Long-Term Outcomes

The mortality risk in VAD is closely tied to the severity of deficiency and the presence of complications. Children with advanced VAD are at particularly high risk of death from infections and systemic complications, emphasizing the need for aggressive treatment and preventive measures in high-risk populations. The prognosis of VAD is highly variable and depends on the stage of deficiency at diagnosis and treatment initiation. Early intervention leads to complete resolution of symptoms and excellent outcomes, while delayed treatment can result in permanent vision loss, increased susceptibility to infections, and significantly higher mortality rates, particularly in children. Public health efforts to improve access to vitamin A supplementation and nutritional education are critical for preventing the severe consequences of VAD.

Complications

Severe vitamin A deficiency (VAD) can lead to a range of serious complications, particularly if left untreated. These include:

- **Permanent Vision Loss or Blindness:** Advanced VAD can cause xerophthalmia, progressing to corneal ulceration, scarring, and keratomalacia, resulting in irreversible blindness [26].
- **Increased Susceptibility to Infections:** VAD compromises mucosal integrity and immune function, making individuals more prone to gastrointestinal, pulmonary, and urinary tract infections.
- **High Mortality:** In children, VAD is associated with significantly increased mortality rates, particularly in those with advanced ocular manifestations such as night blindness, Bitot spots, or keratomalacia [26].

Deterrence and Patient Education

Preventing VAD requires a multifaceted approach, focusing on dietary adequacy, supplementation, and public health interventions.

In Resource-Rich Regions

- A **well-balanced, nutrient-rich diet** is typically sufficient to prevent VAD in healthy individuals. Foods such as dark leafy greens, orange-colored vegetables, dairy products, liver, and fish are excellent sources of vitamin A.
- Many staple foods in these regions are **fortified with vitamin A**, ensuring adequate intake even for those with limited dietary diversity.

In Resource-Poor Regions

- **Large-Scale Supplementation Programs:** The World Health Organization (WHO) recommends high-dose vitamin A supplementation (VAS) at regular intervals for children and at-risk populations. These programs have been implemented globally to reduce VAD prevalence [50].
- **Nutritional Education:** Public health initiatives often include education on **breastfeeding, dietary diversity**, and recognizing **symptoms of VAD**. However, the success of these programs varies by region [51][52].
- **Food Fortification and Biofortification:** Efforts are increasingly focusing on addressing the root causes of VAD through food fortification and biofortification. Staple foods such as rice, wheat, potatoes, and soybeans have been genetically modified to increase their vitamin A content. These biofortified crops are both economically viable and agriculturally sustainable, offering a long-term solution to VAD [53][54][55][56][57][58].

Enhancing Healthcare Team Outcomes

Effective management and prevention of VAD require collaboration among an **interprofessional healthcare team**:

- **Primary Care Physicians:** Often the first point of contact, they coordinate care, provide preventive education, and manage mild cases of VAD.
- **Dietitians and Nutritionists:** Play a key role in educating patients about dietary sources of vitamin A and ensuring adequate nutrition.
- **Pharmacists:** Assist in managing supplementation regimens and monitoring for potential toxicity.
- **Specialists:**
 - **Gastroenterologists:** Manage VAD in patients with malabsorptive conditions such as inflammatory bowel disease (IBD) or pancreatic insufficiency.
 - **Bariatric Surgeons:** Address VAD in post-bariatric surgery patients, who are at high risk due to fat malabsorption.
 - **Transplant Surgeons:** Manage VAD in patients with chronic liver disease or cirrhosis.
 - **Ophthalmologists:** Evaluate and treat VAD-related ocular complications, such as xerophthalmia and keratomalacia [59].

In Resource-Limited Settings

- **Public Health Nurses:** Deliver high-dose VAS to remote areas and provide education on VAD prevention.
- **Governments and International Organizations:** Fund and organize supplementation, fortification, and educational programs to address VAD on a population level [34].

Vitamin A deficiency remains a significant global health challenge, particularly in resource-poor regions. While VAD is simple to diagnose and treat, its complications can be severe, including blindness, increased infection susceptibility, and high mortality. Prevention and management require a collaborative approach involving healthcare providers, public health initiatives, and community education. Efforts to address VAD through supplementation, food fortification, and biofortification are critical for reducing its global burden and improving health outcomes worldwide.

Role of Nutrition Practitioners:

Nutrition practitioners, including dietitians and nutritionists, play a pivotal role in the prevention, management, and treatment of vitamin A deficiency (VAD). Their expertise in dietary assessment, nutritional counseling, and public health interventions is essential for addressing VAD at both individual and population levels. By focusing on dietary adequacy, education, and community outreach, nutrition practitioners contribute significantly to reducing the burden of VAD and its associated complications.

Dietary Assessment and Individualized Counseling

One of the primary roles of nutrition practitioners is to conduct thorough dietary assessments to identify individuals at risk of VAD. This involves evaluating dietary patterns, food intake, and nutritional status to determine whether patients are meeting the recommended dietary allowance (RDA) for vitamin A. For individuals with malabsorptive conditions, such as inflammatory bowel disease (IBD), chronic liver disease, or post-bariatric surgery, nutrition practitioners tailor dietary plans to ensure adequate vitamin A intake despite absorption challenges. They also provide individualized counseling on incorporating vitamin A-rich foods, such as dark leafy greens, orange-colored vegetables, dairy products, liver, and fish, into daily diets. For populations with limited access to animal-based sources of vitamin A, practitioners emphasize the importance of provitamin A carotenoids found in plant-based foods, such as carrots, sweet potatoes, and mangoes.

Public Health and Community Education

In resource-poor regions, where VAD is highly prevalent, nutrition practitioners are instrumental in designing and implementing public health programs aimed at preventing and treating VAD. They

collaborate with governments, non-governmental organizations (NGOs), and international health agencies to deliver nutrition education and promote dietary diversification. For example, they educate communities on the importance of breastfeeding, as breast milk is a critical source of vitamin A for infants, and advocate for the inclusion of vitamin A-rich foods in local diets. Nutrition practitioners also play a key role in supplementation programs, ensuring that high-risk populations, such as children and pregnant women, receive the WHO-recommended doses of vitamin A. By conducting workshops and community outreach initiatives, they raise awareness about the signs and symptoms of VAD, such as night blindness and xerophthalmia, and encourage early intervention.

Food Fortification and Biofortification Initiatives

Nutrition practitioners are at the forefront of efforts to address VAD through food fortification and biofortification. They work with food scientists, agricultural experts, and policymakers to develop and promote fortified staple foods, such as rice, wheat, and cooking oils, enriched with vitamin A. These fortified foods provide a sustainable and cost-effective solution to VAD, particularly in regions where dietary diversity is limited. Additionally, nutrition practitioners advocate for the adoption of biofortified crops, such as orange-fleshed sweet potatoes and golden rice, which are genetically modified to contain higher levels of provitamin A carotenoids. By promoting these innovations, they help bridge the gap between agricultural production and nutritional needs, ensuring that vulnerable populations have access to nutrient-rich foods.

Monitoring and Evaluation

Nutrition practitioners are responsible for monitoring and evaluating the effectiveness of interventions aimed at reducing VAD. This includes tracking the nutritional status of individuals and populations, assessing the impact of supplementation programs, and evaluating the adoption of fortified and biofortified foods. By analyzing data and outcomes, they identify gaps in existing programs and recommend evidence-based strategies to improve their efficacy. For instance, they may advocate for integrating VAD prevention into broader maternal and child health programs or strengthening partnerships with local farmers to promote biofortified crops.

Collaboration with Healthcare Teams

Nutrition practitioners work closely with other healthcare professionals, including physicians, nurses, and specialists, to provide comprehensive care for individuals with VAD. They contribute to interprofessional teams by offering nutritional insights and developing tailored dietary plans for patients with conditions that increase the risk of VAD, such as cystic fibrosis, chronic pancreatitis, or liver cirrhosis. In clinical settings, they collaborate with ophthalmologists to manage VAD-related ocular complications and with gastroenterologists to address malabsorptive disorders. Their role is particularly critical in post-bariatric surgery patients, who require specialized nutritional support to prevent deficiencies due to altered digestion and absorption. Nutrition practitioners are indispensable in the fight against vitamin A deficiency. Through dietary assessment, public health education, food fortification, and collaboration with healthcare teams, they address the root causes of VAD and implement sustainable solutions to improve nutritional outcomes. Their efforts not only prevent and treat VAD but also contribute to broader public health goals, such as reducing childhood mortality, improving maternal health, and enhancing overall well-being. By leveraging their expertise, nutrition practitioners play a vital role in ensuring that individuals and communities have access to the nutrients they need to thrive.

Conclusion:

Vitamin A deficiency (VAD) remains a critical public health challenge, particularly in resource-poor regions where access to nutrient-rich foods is limited. This review underscores the importance of vitamin A in maintaining vision, immune function, and overall health, and highlights the severe consequences of its deficiency, including blindness, increased susceptibility to infections, and high mortality rates, especially among children and pregnant women. The etiology of VAD varies between developing and developed regions, with poor nutrition and recurrent infections being primary drivers in low-income settings, while

malabsorptive disorders and chronic diseases are more common in high-income countries. The clinical presentation of VAD ranges from subclinical deficiency to advanced stages characterized by night blindness, Bitot spots, and corneal ulceration. Early diagnosis and treatment are crucial to prevent irreversible complications. Serum retinol levels are widely used for diagnosis, though they have limitations, and liver biopsy remains the most accurate method for assessing total body stores. Treatment primarily involves vitamin A supplementation (VAS), with dosing regimens tailored to age, severity, and regional guidelines. In resource-poor regions, universal supplementation programs have significantly reduced VAD-related morbidity and mortality. Prevention strategies are equally important and include dietary diversification, food fortification, and biofortification of staple crops. These approaches address the root causes of VAD by improving access to vitamin A-rich foods and ensuring sustainable nutritional solutions. Public health education plays a vital role in raising awareness about VAD and promoting breastfeeding, dietary diversity, and early intervention. The role of nutrition practitioners is indispensable in combating VAD. Through dietary assessment, individualized counseling, and community education, they help identify at-risk individuals and implement effective interventions. Collaboration among healthcare professionals, including primary care physicians, dietitians, pharmacists, and specialists, is essential for comprehensive management. In conclusion, VAD is a preventable condition with far-reaching health implications. Addressing it requires a coordinated effort involving supplementation, dietary interventions, and public health initiatives. By leveraging the expertise of nutrition practitioners and fostering interprofessional collaboration, we can reduce the global burden of VAD and improve health outcomes for vulnerable populations worldwide.

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الملخص:

الخلفية: نقص فيتامين أ (VAD) هو قضية صحية عالمية كبيرة، يؤثر بشكل خاص على الأطفال والنساء الحوامل في المناطق ذات الدخل المنخفض. يعتبر فيتامين أ ضروريًا للرؤية، وظيفة المناعة، وتطور الخلايا، وقد يؤدي نقصه إلى مضاعفات خطيرة، بما في ذلك العمى، زيادة القابلية للعدوى، وارتفاع معدلات الوفيات. بينما يعد نقص فيتامين أ نادرًا في الدول المتقدمة، إلا أنه لا يزال منتشرًا في المناطق الفقيرة بسبب عدم كفاية المدخول الغذائي والالتهابات المتكررة. تم تنفيذ تدخلات الصحة العامة، مثل المكملات الغذائية، تدعيم الأغذية، والتغذية الحيوية، لمعالجة هذه المشكلة.

الهدف: تهدف هذه المراجعة إلى تزويد المهنيين الصحيين وأخصائي التغذية بفهم محدث لنقص فيتامين أ، بما في ذلك أسبابه، انتشاره، آلية حدوثه، الأعراض السريرية، التقييم التشخيصي، العلاج، واستراتيجيات الوقاية.

المنهجية: تقوم المراجعة بتلخيص الأدبيات الحالية والإرشادات المتعلقة بنقص فيتامين أ، مع التركيز على انتشاره العالمي، عوامل الخطر، المظاهر السريرية، وإدارته. تسلط الضوء على أدوار تقييم النظام الغذائي، المكملات الغذائية، وتدخلات الصحة العامة في معالجة نقص فيتامين أ.

النتائج: يؤثر نقص فيتامين أ بشكل غير متناسب على الأطفال دون الخامسة والنساء الحوامل في الدول النامية، مما يساهم في معدلات مراضة ووفيات عالية. تشمل الأعراض المبكرة العشى الليلي وجفاف الملتحمة، والتي يمكن أن تتطور إلى عمى دائم إذا لم يتم علاجها. يتم استخدام مستويات الريتينول في الدم بشكل شائع للتشخيص، على الرغم من أن خزعة الكبد تظل المعيار الذهبي. يتضمن العلاج مكملات فيتامين أ (VAS)، مع تحديد الجرعات بناءً على العمر، شدة النقص، والإرشادات الإقليمية. تشمل استراتيجيات الوقاية تنوع النظام الغذائي، تدعيم الأغذية، والتغذية الحيوية للمحاصيل الأساسية.

الاستنتاج: نقص فيتامين أ هو حالة يمكن الوقاية منها ولكنها قد تؤدي إلى عواقب وخيمة إذا تركت دون علاج. تتطلب الإدارة الفعالة نهجًا متعدد الجوانب، بما في ذلك المكملات الغذائية، التثقيف الغذائي، ومبادرات الصحة العامة. يعد التعاون بين المهنيين الصحيين، أخصائي التغذية، وصناع السياسات أمرًا ضروريًا للحد من العبء العالمي لنقص فيتامين أ.

الكلمات المفتاحية: نقص فيتامين أ، جفاف الملتحمة، المكملات الغذائية، تدعيم الأغذية، التغذية الحيوية، الصحة العامة.