



Clinical and Nutritional Implications of Vitamin A-An Updated Review for Dietitian and Nutritionists.

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Abstract

Background: Vitamin A encompasses retinol, retinyl esters, and provitamin A carotenoids that underpin vision, epithelial integrity, immune modulation, and gene regulation through retinal and retinoic acid.

Aim: To synthesize contemporary evidence on clinical indications, mechanisms, pharmacokinetics, dosing, population-specific considerations, adverse effects, and monitoring strategies for vitamin A to inform dietitians, nutritionists, and clinicians.

Methods: Narrative review of established guidance and summarized evidence as presented in the manuscript, including FDA-approved and off-label uses, WHO grading of deficiency manifestations, pediatric and maternal considerations, toxicity profiles, drug interactions, and monitoring parameters.

Results: Vitamin A deficiency remains a global public-health challenge, with supplementation reducing childhood morbidity and mortality, and serving key roles in xerophthalmia, severe malnutrition, measles, and select neonatal indications. Parenteral therapy is reserved for malabsorption or intolerance to oral dosing. Mechanistically, vitamin A supports phototransduction and RAR-mediated transcription, while hepatic storage and biliary elimination govern its pharmacokinetics. Risk-benefit varies in pregnancy and hepatic/renal impairment. Toxicity spans acute to chronic syndromes, including hepatotoxicity and teratogenicity; interactions with agents such as orlistat and oral contraceptives warrant caution. Monitoring relies primarily on serum retinol (as a proxy for hepatic stores) and targeted labs when derivatives like isotretinoin are used.

Conclusion: Judicious, evidence-based vitamin A use—prioritizing dietary sources, targeted supplementation in high-risk settings, age- and condition-appropriate dosing, and vigilant monitoring—maximizes benefit while minimizing toxicity. Interprofessional coordination is essential to optimize outcomes.

Keywords: Vitamin A; retinol; β -carotene; xerophthalmia; measles; pediatric supplementation; pregnancy; hepatotoxicity; pharmacokinetics; monitoring; retinoic acid receptors.

Introduction

Vitamin A comprises a group of fat-soluble compounds, including retinol, retinyl palmitate, and β -carotene, which are essential for numerous physiological processes. These metabolites play a critical role in vision, particularly in the formation of the visual pigment rhodopsin, cellular differentiation, the maintenance of epithelial integrity, and immune

system function.[1] Adequate intake of vitamin A is therefore necessary to preserve the structural and functional integrity of multiple organ systems. Dietary vitamin A exists in two primary forms. Preformed vitamin A, consisting of retinol and retinyl esters, is primarily obtained from animal-based foods such as liver, dairy products, fish, and eggs. Provitamin A carotenoids, predominantly β -carotene, are found in

brightly colored fruits and vegetables including carrots, sweet potatoes, and leafy greens. Both forms require metabolic conversion after absorption; retinol and carotenoids are transformed into retinal and retinoic acid, the biologically active forms responsible for vision, gene expression, and epithelial maintenance.[1] Vitamin A deficiency results in significant pathological changes, particularly the replacement of normal epithelial tissue with keratinized stratified epithelium. This process affects ocular tissues, periocular glands, respiratory epithelium, the gastrointestinal tract, and the genitourinary tract.[2] Clinically, deficiency can manifest as night blindness, conjunctival xerosis, Bitot spots, corneal xerosis, keratomalacia, and permanent corneal scarring. The World Health Organization estimates that approximately three million children develop clinical manifestations of vitamin A deficiency annually, whereas toxicity is comparatively rare, with around 200 cases reported each year.[3]

Globally, vitamin A deficiency represents a major public health challenge, particularly in low-income countries where malnutrition is prevalent. Supplementation programs have been shown to significantly reduce child morbidity and mortality from preventable infectious diseases, making vitamin A supplementation one of the most cost-effective interventions in public health. Vitamin A is indicated for the treatment of xerophthalmia, severe malnutrition, and as a prophylactic measure in pregnant women living in regions where deficiency is endemic.[4] Measles treatment also benefits from vitamin A supplementation, as it reduces the risk of severe complications, including pneumonia and ocular damage. While some research has explored vitamin A's potential role in cancer prevention, evidence remains inconclusive. In populations with sufficient dietary vitamin A, supplementation has not demonstrated additional protective effects. However, in vitamin A-deficient populations, such as malnourished individuals or chronic tobacco users, supplementation may confer a reduced risk of certain malignancies.[5] Additionally, a meta-analysis highlighted that individuals with tuberculosis often present with low levels of vitamin A and D, suggesting a potential role in immune support and recovery.[6] The WHO grading system for pediatric eye signs of vitamin A deficiency stratifies the severity of xerophthalmia. Initial manifestations include night blindness (XN) and conjunctival xerosis (X1A), progressing to Bitot spots (X1B) and corneal xerosis (X2). Corneal ulceration (X3A and X3B) indicates advanced deficiency, with X3B representing extensive keratomalacia, which may result in corneal perforation. Permanent complications, including corneal scarring (XS), reflect the severe consequences of prolonged deficiency and underscore the importance of early intervention. Vitamin A supplementation remains a cornerstone in preventing

blindness and supporting overall health in at-risk populations. Its clinical indications extend from prophylaxis in deficiency-prone regions to therapeutic applications in malnourished children, individuals with measles, and patients presenting with ocular manifestations of deficiency. The integration of dietary, therapeutic, and public health approaches ensures both the prevention and treatment of deficiency, reducing the global burden of associated morbidity and mortality.

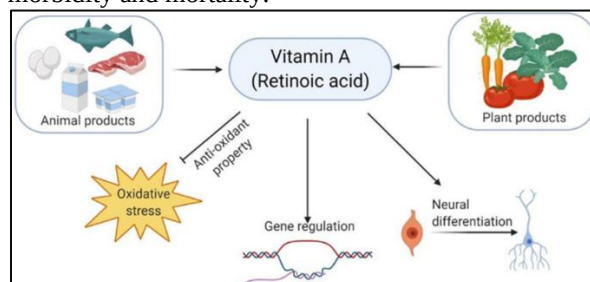


Fig. 1: Vitamin A supplement.

FDA-Approved Indications

The United States Food and Drug Administration (FDA) has approved the parenteral administration of vitamin A primarily for the treatment of vitamin A deficiency in individuals who are unable to receive oral supplementation. This route is particularly indicated in patients experiencing persistent vomiting, those in preoperative or postoperative states, or individuals with gastrointestinal malabsorption syndromes that impede the absorption of orally administered vitamin A.[7][8] Parenteral vitamin A provides a reliable and rapid means of restoring physiological vitamin A levels, ensuring that critical cellular and systemic functions—such as vision, epithelial maintenance, and immune defense—are preserved even in situations where oral intake is not feasible.

Off-Label Uses

Beyond its FDA-approved indications, parenteral and supplemental vitamin A have been investigated for several off-label applications with potential clinical benefits. One of the most studied off-label uses is in infants with extremely low birth weight. These neonates are at high risk of developing bronchopulmonary dysplasia (BPD), a chronic lung disease associated with prolonged oxygen therapy and mechanical ventilation. Repeated parenteral doses of vitamin A have been shown to reduce the incidence and severity of BPD, though the procedure is associated with discomfort and challenges in administration. Research continues to evaluate whether enteral supplementation using water-soluble vitamin A formulations might offer similar protective benefits while minimizing procedural stress for neonates.[9] Vitamin A also appears to influence immune modulation, particularly in relation to autoimmune conditions. Evidence suggests that deficiency impairs the function of helper T cells, which are central to the autoimmune response in type

1 diabetes (T1D). Serum vitamin A levels are frequently lower in patients with T1D, indicating a potential contributory role in disease pathophysiology and progression. While the exact mechanisms remain under investigation, this association highlights a possible therapeutic or preventive role for vitamin A in the management of T1D, pending further research.[10]

In addition to autoimmune considerations, vitamin A deficiency significantly impacts infectious disease outcomes. Deficiency compromises normal immune function, reducing resistance to pathogens such as the measles virus, which remains a major cause of childhood morbidity and mortality globally. Megadoses of vitamin A, such as 200,000 IU administered over two days, have been demonstrated to reduce measles-related mortality and improve clinical outcomes in children affected by the disease.[3] Furthermore, vitamin A deficiency contributes to ocular surface disorders. According to the American Academy of Ophthalmology, insufficient vitamin A is a recognized risk factor for dry eye disease, in addition to its role in xerophthalmia and other visual impairments. Supplementation, whether for preventive or therapeutic purposes, can restore ocular epithelial integrity, maintain tear film homeostasis, and reduce the risk of keratinization and corneal complications.[11] Collectively, the FDA-approved and off-label applications of vitamin A highlight its broad clinical utility, ranging from the management of classic deficiency states to potential interventions in neonatal respiratory disease, autoimmune disorders, infectious diseases, and ocular health. These indications underscore the critical role of vitamin A in both systemic physiology and specialized clinical contexts, with ongoing research aimed at expanding its evidence-based applications.

Mechanism of Action

Vitamin A undergoes complex metabolic processing that enables it to perform its critical biological functions. In the liver, retinol is esterified into retinyl esters and stored in hepatic stellate cells, providing a reserve that can be mobilized when tissue demand increases.[1] Upon release, retinol and dietary provitamin A, such as β -carotene, are metabolized into active derivatives: retinal and retinoic acid. Retinal is primarily involved in vision, serving as the chromophore in rhodopsin and cone pigments, enabling phototransduction and low-light vision. Retinoic acid acts as a transcriptional regulator, binding to nuclear receptors of the retinoic acid receptor (RAR) family to modulate gene expression across multiple tissues.[12][13][14] Through these mechanisms, vitamin A exerts effects on cellular differentiation, proliferation, and morphogenesis. Retinoic acid influences the transcription of genes involved in epithelial tissue maintenance, supporting the regeneration of mucosal surfaces and the integrity of organs such as the respiratory tract, gastrointestinal tract, and genitourinary system. These actions are

particularly critical in maintaining barrier function against pathogens. Vitamin A also modulates the immune system. It is essential for the development and function of helper T cells and B cells, facilitating adaptive immunity. Additionally, it enhances innate immunity by supporting neutrophil, macrophage, and natural killer cell activity. This immune modulation reduces susceptibility to infectious diseases and enhances pathogen clearance. Furthermore, vitamin A acts as an antioxidant, protecting cells from oxidative stress by neutralizing free radicals. This property preserves DNA integrity and reduces the risk of mutation. Deficiency in vitamin A impairs these protective and regulatory mechanisms, contributing to increased susceptibility to infection, impaired tissue regeneration, and, potentially, neoplastic transformation and carcinogenesis.[5] Overall, the metabolic conversion of vitamin A into retinal and retinoic acid integrates visual function, immune regulation, epithelial maintenance, and gene expression, establishing vitamin A as an essential micronutrient for overall health and cellular homeostasis.

Pharmacokinetics

Vitamin A exhibits complex pharmacokinetic properties that are essential for its biological functions. Absorption occurs predominantly in the small intestine, where dietary retinol and provitamin A carotenoids, such as β -carotene, are processed. Efficient absorption requires the presence of bile salts, pancreatic lipase, and dietary fat, which facilitate the formation of micelles that enable retinoid uptake by enterocytes. Within the intestinal cells, β -carotene is enzymatically cleaved into retinol, which can then be esterified and incorporated into chylomicrons for transport.[15] Following absorption, vitamin A is distributed systemically through the lymphatic system. Chylomicrons transport retinol and retinyl esters to the liver, which serves as the primary storage site, holding approximately 70% of total body vitamin A reserves. Additional storage occurs in adipose tissue and other organs, providing a buffer against periods of dietary deficiency. Retinol bound to retinol-binding protein (RBP) facilitates transport from the liver to peripheral tissues, where it is converted into its active metabolites, retinal and retinoic acid, to support vision, gene expression, and cellular differentiation. Metabolism of vitamin A is primarily hepatic, with retinol stored as retinyl palmitate in stellate cells. These hepatic stores are substantial, capable of meeting physiological demands for up to two years under normal conditions. In tissues, retinol is oxidized to retinal and retinoic acid to exert its functional effects. Elimination of vitamin A occurs predominantly via biliary excretion into the intestines, with fecal elimination representing the major route. Minor amounts of vitamin A and its metabolites are excreted in urine. The long-term storage, regulated release, and biliary excretion contribute to maintaining

stable plasma concentrations of vitamin A under variable dietary intake, ensuring sustained availability for critical biological processes.

Administration

Vitamin A is available in multiple formulations and strengths to accommodate various clinical needs. Oral preparations include capsules containing 7,500 U, 8,000 U, 10,000 U, and 25,000 U, as well as tablets in doses of 10,000 U and 15,000 U. For parenteral administration, vitamin A palmitate is supplied as an injectable solution at 50,000 USP U/mL, equivalent to 15 mg of retinol per milliliter. These formulations allow flexibility in addressing both prophylactic and therapeutic requirements.[1] Administration routes depend on the patient's condition and the urgency of treatment. Oral administration is preferred for most patients, particularly when dietary intake can be enhanced with meals containing sufficient fat, as the lipophilic nature of vitamin A improves its intestinal absorption. Intramuscular injection is reserved for patients unable to tolerate oral intake due to vomiting, malabsorption syndromes, or preoperative or postoperative states. Parenteral administration ensures rapid delivery and bioavailability in critical situations, such as severe deficiency or acute xerophthalmia. For adults with vitamin A deficiency, initial therapy typically involves high-dose administration to rapidly replenish body stores and correct clinical manifestations. The recommended initial dose is 100,000 U daily for three consecutive days, which is followed by 50,000 U daily for two weeks. This regimen is designed to restore plasma and tissue vitamin A levels efficiently. After the initial high-dose course, maintenance therapy is recommended to prevent recurrence of deficiency. Oral multivitamins containing 10,000 to 20,000 U of vitamin A daily for approximately two months help sustain adequate vitamin A levels and support recovery of epithelial, visual, and immune functions. Regular monitoring during treatment ensures safety and allows adjustments for patient-specific needs, including comorbid conditions or risk factors for hypervitaminosis A.

Specific Patient Populations

Vitamin A use requires careful consideration in certain patient populations due to altered physiology, potential toxicity, and developmental implications. In individuals with hepatic impairment, no specific dosage adjustments are provided by the manufacturer. However, given that the liver is the primary site of vitamin A storage, metabolism, and conversion to its active forms, clinicians should monitor patients with hepatic disease for signs of accumulation or hypervitaminosis. Liver dysfunction may reduce the capacity to store retinol as retinyl esters and impair mobilization, potentially increasing the risk of toxicity even with standard doses. Laboratory monitoring of liver function and clinical observation are recommended in these cases.

Similarly, no dosage adjustment guidelines are provided for patients with renal impairment. Vitamin A is primarily excreted through bile into the gastrointestinal tract, with minimal renal elimination. Although renal dysfunction does not directly alter vitamin A clearance, it may exacerbate complications associated with hypervitaminosis, such as hypercalcemia or metabolic imbalances. Caution and regular monitoring are advised when prescribing high doses in this population.

Pregnancy presents unique challenges regarding vitamin A use. Excessive intake can be teratogenic, particularly during early gestation. A documented case describes a 36-year-old pregnant woman who developed vitamin A toxicity after consuming fish liver, resulting in miscarriage, highlighting the potential fetal risks associated with high intake.[16] Maternal ingestion exceeding 6,000 U daily has not been established as safe and may cause congenital malformations affecting the central nervous system, eyes, palate, and urogenital system.[17] Consequently, supplementation beyond the recommended dietary allowance is contraindicated in women who are pregnant or may become pregnant. Despite this, vitamin A is essential for fetal development, particularly for ocular integrity and epithelial differentiation. In regions with prevalent deficiency, targeted supplementation remains necessary to prevent maternal and infant morbidity, including night blindness. Routine supplementation is generally not recommended in developed countries where vitamin A deficiency is rare.[18] During lactation, vitamin A continues to play a critical role. Infants are born with limited liver stores of vitamin A, even when maternal intake is adequate. Breast milk, particularly colostrum, provides a crucial source of retinol and retinyl esters, supporting neonatal vitamin A reserves and ensuring sufficient intake during periods of limited dietary availability. The concentration of vitamin A in breast milk varies regionally, influenced by dietary intake and the presence of carotenoids such as β -carotene, α -carotene, and β -cryptoxanthin. Maternal vitamin A status directly affects the quantity of retinol and retinyl esters incorporated into milk via chylomicrons after lipolysis, underscoring the importance of adequate maternal nutrition during lactation. While vitamin A requirements increase during breastfeeding, limited data exist regarding the precise impact on maternal stores, assuming that enhanced maternal intake supports adequate milk concentrations and infant health.[19] In conclusion, clinicians must individualize vitamin A administration for patients with hepatic or renal impairment, women of childbearing age, and lactating mothers, balancing the prevention of deficiency with the risk of toxicity. Careful monitoring, adherence to recommended dietary allowances, and education regarding safe

intake are essential to optimize maternal and infant outcomes.

Pediatric Patients

Vitamin A supplementation has a well-established role in pediatric health, particularly in children aged 6 months to 5 years, where deficiency remains a major public health concern. Systematic reviews demonstrate that providing vitamin A to this age group significantly reduces morbidity and mortality, especially in regions where deficiency is endemic. Supplementation has been shown to prevent or reduce the severity of diarrhea, measles, and night blindness, highlighting its essential role in supporting immune function, epithelial integrity, and visual health in young children.[20] These effects are particularly important in low- and middle-income countries, where vitamin A deficiency is a leading contributor to childhood morbidity. By reducing the incidence of severe infections and promoting proper growth and development, vitamin A supplementation has a measurable impact on pediatric survival rates and long-term health outcomes. For clinical management, parenteral vitamin A administration is indicated when oral supplementation is not feasible due to vomiting, malabsorption, or severe illness. Product labeling recommends specific regimens based on age. For children aged 1 to 8 years, a parenteral dose of 17,500 to 35,000 U daily for 10 days is advised, followed by oral multivitamin supplementation providing 5,000 to 10,000 U daily for two months. In infants, parenteral administration ranges from 7,500 to 15,000 U daily for 10 days, with a similar oral follow-up. These regimens aim to rapidly restore hepatic vitamin A stores, correct deficiency symptoms, and maintain sufficient levels during the recovery period. Professional guidance from the American Society for Parenteral and Enteral Nutrition (ASPEN) supports these recommendations and emphasizes the importance of tailored supplementation in pediatric patients receiving parenteral nutrition. These guidelines, as highlighted in the 2024 Parenteral Nutrition Product Shortage Recommendations, ensure that infants and children at risk of deficiency receive adequate vitamin A, even in clinical settings where oral administration is impractical. In older adults, maintaining adequate vitamin A intake is important to support immune function, vision, and cellular differentiation. Recommended intake for individuals aged 51 years and older is 900 mcg Retinol Activity Equivalents (RAE) per day for males and 700 mcg RAE per day for females. While deficiency is less common in older populations in developed countries, ensuring sufficient intake helps prevent age-related visual impairment and supports overall health. Nutritional planning, including dietary sources and supplementation when necessary, is recommended to maintain these levels, particularly in older adults with limited dietary intake or malabsorption issues. In both pediatric and older populations, careful attention to dosing and administration is essential to

avoid toxicity while ensuring adequate vitamin A status.

Adverse Effects

Vitamin A, while essential for vision, immune function, and cellular differentiation, can cause significant adverse effects when ingested in excessive amounts. Both natural and synthetic forms of vitamin A are capable of producing toxicity, with hypervitaminosis A most commonly arising from supplement misuse rather than from routine dietary intake. Consumption of animal livers, particularly cod liver, or chronic use of vitamin A-containing preparations can also contribute to toxic accumulation. The threshold for toxicity varies between individuals, influenced by factors such as age, body weight, and hepatic function, as vitamin A is primarily stored in the liver. Acute vitamin A toxicity is generally associated with a single ingestion of 25,000 IU/kg or more. Clinical manifestations include gastrointestinal disturbances such as nausea, vomiting, and diarrhea, along with neurological symptoms like dizziness, lethargy, and drowsiness. Increased intracranial pressure may also develop, particularly in children, presenting with headache, irritability, or bulging fontanelles. Dermatological effects, including erythema, pruritus, and desquamation, may be observed. Chronic vitamin A toxicity occurs with prolonged ingestion, typically exceeding 4,000 IU/kg per day for six months or longer. Symptoms may include low-grade fever, persistent headache, fatigue, anorexia, and gastrointestinal complaints. Hepatosplenomegaly, anemia, hypercalcemia, subcutaneous edema, nocturia, musculoskeletal discomfort, and joint pain are common systemic manifestations. Dermatological changes, such as dry, yellowed skin, alopecia, and photosensitivity, are frequent. Chronic toxicity can also interfere with bone metabolism, increasing the risk of fractures and skeletal abnormalities. Vitamin A is highly teratogenic during pregnancy. Retinoids influence the expression of homeobox genes, such as Hoxb-1, which regulate embryonic axial patterning. Excessive maternal intake can lead to craniofacial, cardiac, and central nervous system malformations. Supplementation should be avoided during pregnancy except in regions with endemic vitamin A deficiency, and doses should not exceed 10,000 IU daily to mitigate fetal risk.[21] Mild adverse effects may occur when vitamin A is administered alongside immunizations, including loose stools, headache, irritability, fever, nausea, and vomiting. These reactions are typically self-limiting and resolve within 24 to 48 hours.

Drug-Drug Interactions

Vitamin A absorption is influenced by coadministration of certain drugs. Orlistat, a gastrointestinal lipase inhibitor, reduces fat absorption and consequently decreases the uptake of fat-soluble vitamins, including vitamin A. Patients should be counseled to separate vitamin A supplementation from orlistat administration by at least two hours to

optimize absorption.[22] Interactions have also been reported with oral contraceptive pills. Women using oral contraceptives may exhibit elevated plasma vitamin A levels, warranting caution in supplementation to avoid potential toxicity.[23] Overall, careful attention to dosing, patient age, underlying health conditions, pregnancy status, and concomitant medications is essential to prevent adverse effects and optimize the safe use of vitamin A.

Contraindications

Vitamin A administration is contraindicated in pregnant women due to its established teratogenic potential. Excessive vitamin A intake during pregnancy, particularly in the first trimester, can result in severe fetal malformations affecting the craniofacial structure, central nervous system, heart, and urogenital tract. Similarly, breastfeeding patients should exercise caution with vitamin A supplementation, as excessive maternal intake may alter the concentration in breast milk, posing potential risks to the neonate. Individuals with a known hypersensitivity to vitamin A or its derivatives are also contraindicated from receiving the vitamin, as allergic reactions ranging from mild rash to severe anaphylaxis may occur. Caution is recommended when prescribing vitamin A to patients with pre-existing hepatic or renal impairment. The liver is the primary storage site for vitamin A, and hepatic dysfunction can increase the risk of accumulation and toxicity. Similarly, impaired renal excretion may exacerbate hypervitaminosis A. Chronic alcohol use is another factor that predisposes patients to hepatotoxicity, necessitating careful monitoring when vitamin A supplementation is indicated. Patients with acne vulgaris should also be evaluated cautiously, as high doses of vitamin A derivatives, such as retinoids, can exacerbate cutaneous symptoms or increase the risk of systemic adverse effects.[24]

Warnings and Precautions

High-dose vitamin A supplementation, particularly β -carotene, has been associated with potential long-term risks. Meta-analyses suggest an increased incidence of lung cancer in smokers and elevated cardiovascular mortality among certain populations exposed to high levels of β -carotene. Hepatotoxicity, manifesting as elevated liver enzymes or structural liver changes, may also result from chronic excessive intake. Clinicians should weigh the benefits of supplementation against these potential risks and consider baseline liver function testing and ongoing monitoring for patients receiving high doses.[25] Emerging evidence indicates a possible link between vitamin A and idiopathic intracranial hypertension, though causality remains under investigation. Symptoms such as headache, visual disturbances, and papilledema warrant vigilance in patients receiving high-dose vitamin A therapy. Further research is needed to clarify the relationship and guide clinical recommendations.[26] Overall,

vitamin A should be prescribed judiciously, with careful assessment of patient-specific risk factors, comorbidities, and potential drug interactions to ensure safety and efficacy.

Monitoring

Monitoring vitamin A status and safety during supplementation or treatment with vitamin A derivatives is critical, as both deficiency and excess carry significant clinical consequences. Serum retinol measurement is commonly used to assess vitamin A status, but it does not always correlate precisely with liver retinyl ester stores, which are the primary reservoir for this fat-soluble vitamin. Nevertheless, serum retinol levels remain a practical clinical tool because they decline when liver stores become depleted, providing an indirect but useful marker of deficiency.[27] Clinicians should interpret these results in the context of dietary intake, underlying illnesses, and ongoing supplementation. When using vitamin A derivatives such as isotretinoin, which is indicated for severe acne, laboratory monitoring is recommended to ensure safety. Isotretinoin is associated with potential hepatotoxicity and dyslipidemia, requiring baseline evaluation of liver function tests and lipid panels. Repeated monitoring was traditionally recommended weekly or biweekly until a clinical response is achieved. However, recent evidence from meta-analyses indicates that most significant elevations in triglycerides and cholesterol occur within the first eight weeks of therapy, with minimal further change thereafter. Liver function test abnormalities are uncommon and, when they occur, are rarely clinically significant. These findings suggest that routine monthly laboratory testing may be unnecessary for patients receiving standard oral isotretinoin dosages, allowing for more individualized monitoring based on patient risk factors and early biochemical responses.[28] Overall, effective monitoring of vitamin A therapy requires balancing the risks of toxicity with the need to correct deficiency or achieve therapeutic outcomes. Regular clinical evaluation, targeted laboratory assessments, and careful patient counseling on dietary intake and supplementation practices are essential. By integrating these strategies, healthcare providers can minimize adverse effects while maximizing the therapeutic benefits of vitamin A and its derivatives.

Toxicity

Vitamin A toxicity, or hypervitaminosis A, can manifest through a spectrum of clinical signs and laboratory abnormalities. Patients may present with nonspecific symptoms such as abdominal pain, nausea, vomiting, headache, and facial flushing. Chronic or severe cases may exhibit more pronounced neurologic symptoms, including irritability, lethargy, and increased intracranial pressure. Dermatologic manifestations, including dry, scaly skin, alopecia, and desquamation, are also commonly observed. Laboratory investigations often reveal mild anemia,

thrombocytopenia, elevated liver enzymes, and significantly increased serum retinol or retinyl ester levels. Although the diagnosis is primarily clinical, laboratory measurements of serum retinol provide supportive confirmation, particularly when the history suggests excessive intake. A detailed dietary and supplementation history is essential to identify sources of overconsumption, which may include high-dose supplements, fortified foods, or vitamin A-rich animal products, such as liver. Management of vitamin A toxicity begins with the immediate cessation of all vitamin A sources. Most patients experience gradual resolution of symptoms and normalization of laboratory values within several weeks. Supportive care is critical, particularly in severe cases. For patients with increased intracranial pressure, therapeutic interventions such as lumbar puncture or osmotic diuretics like mannitol may be indicated to reduce intracranial pressure and prevent complications. Hypercalcemia secondary to vitamin A excess requires aggressive management with intravenous fluids to promote renal excretion, alongside agents such as calcitonin or corticosteroids when indicated. Continuous monitoring of liver function and hematologic parameters ensures that complications such as hepatotoxicity or thrombocytopenia are addressed promptly. Case reports have documented that discontinuation of vitamin A leads to the resolution of cutaneous symptoms, normalization of serum retinol levels, and improvement in hematologic and hepatic abnormalities. These observations underscore the importance of cautious dosing, particularly in populations at risk for inadvertent overconsumption, such as children receiving routine supplementation or patients self-administering high-dose vitamins. Early recognition, prompt discontinuation of vitamin A, and supportive care remain the cornerstones of management to prevent long-term sequelae associated with hypervitaminosis A.[29]

Enhancing Healthcare Team Outcomes

Optimizing outcomes in the management of vitamin A requires a coordinated, interprofessional approach. Healthcare providers, including clinicians, nurses, dietitians, pharmacists, and public health specialists, must recognize that empirical administration of vitamin A is not recommended. Supplementation should be limited to patients with a clinically diagnosed deficiency, as indiscriminate use can lead to toxicity without providing measurable health benefits. Patient education should emphasize obtaining vitamin A from a balanced diet rich in animal products and colorful fruits and vegetables rather than relying on high-dose supplements, particularly in populations at low risk of deficiency.[30] Pediatricians play a critical role in identifying vitamin A deficiency in children, assessing risk factors, and determining appropriate therapeutic interventions. Early diagnosis and supplementation are particularly important in preventing complications

such as xerophthalmia, night blindness, and impaired immune function. Nutritionists contribute by evaluating dietary patterns, designing individualized meal plans, and guiding safe supplementation to ensure adequate intake while avoiding excess. Ophthalmologists provide specialized assessment of ocular manifestations and monitor for improvements or complications following supplementation, while also remaining vigilant for signs of hypervitaminosis A. Public health professionals develop and implement community-level strategies, including supplementation programs, food fortification, and educational campaigns to prevent deficiency while minimizing the risk of toxicity in vulnerable groups.

Nurses are central to patient safety, as they monitor for both deficiency and toxicity, educate patients and caregivers on appropriate dosing, and assist in administering supplements according to evidence-based protocols. Pharmacists ensure accurate dispensing, verify appropriate dosing, and provide counseling regarding interactions and adverse effects. In cases where neurological symptoms develop, such as increased intracranial pressure or irritability from excessive vitamin A intake, neurologists assess and manage these complications, collaborating closely with the broader care team. Effective communication and collaboration across disciplines are essential. Regular team discussions, shared documentation, and integrated care pathways help identify at-risk patients, coordinate interventions, and monitor outcomes. By maintaining a structured, interprofessional approach, healthcare teams can maximize the benefits of vitamin A supplementation for those in need, minimize the risk of toxicity, and ensure safe, patient-centered care that balances efficacy with safety.

Conclusion:

Vitamin A remains indispensable for visual function, epithelial maintenance, immune competence, and gene regulation, yet its clinical value depends on precise indication, dose, route, and monitoring. Current evidence supports supplementation to prevent blindness and reduce infectious disease morbidity in deficiency-prone settings, including routine pediatric programs and measles management; intramuscular therapy is reserved for patients unable to tolerate or absorb oral formulations. Equally important are context-specific cautions: pregnancy demands strict avoidance of excess due to teratogenicity; hepatic disease, alcohol use, and potential drug interactions elevate toxicity risk; and high-dose or chronic intake can precipitate hepatotoxicity, skeletal effects, and intracranial hypertension. Practical stewardship centers on confirming deficiency, restoring hepatic stores with appropriate loading and maintenance regimens, favoring diet-based strategies when feasible, and tailoring care for infants, children, and older adults. Monitoring with serum retinol (acknowledging its limitations as a surrogate for liver stores) and targeted

laboratory panels—particularly when prescribing retinoids—supports safe therapy. Ultimately, interprofessional collaboration among clinicians, dietitians, pharmacists, nurses, and public-health teams enables targeted interventions that maximize therapeutic gains while minimizing harm, advancing patient- and population-level outcomes in settings where deficiency persists and in specialized clinical scenarios where vitamin A retains unique utility.

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