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The Relationship between Skin Diseases and Nutritional Deficiencies in Children

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ABSTRACT

Background: Nutritional deficiencies are a significant global health issue, particularly in children, as they can impair growth and development. The skin often reflects early signs of nutritional deficiencies, including macronutrient and micronutrient imbalances, which may manifest as specific dermatological symptoms. Early identification of these manifestations is crucial to prevent long-term morbidity and mortality.
Aims: This review aims to explore the relationship between nutritional deficiencies and skin diseases in children, emphasizing clinical features, diagnostic approaches, and effective treatment strategies.
Methods: A comprehensive literature review was conducted using relevant studies and case reports on pediatric dermatological manifestations caused by macronutrient and micronutrient deficiencies.
Discussion: Nutritional deficiencies, including protein-energy malnutrition, essential fatty acid deficiency, and deficiencies of vitamins A, B-complex, C, and K, as well as zinc and copper, present with a wide range of skin manifestations. These include xerosis, dermatitis, hyperkeratosis, hyperpigmentation, and petechiae. Diagnosis involves a combination of dietary history, clinical examination, and laboratory tests to assess nutritional status. Treatment requires nutritional supplementation tailored to the specific deficiency, alongside supportive dermatological care.
Conclusion: Dermatological manifestations are often the first indicators of nutritional deficiencies in children. Early recognition and a multidisciplinary approach to diagnosis and treatment are essential to improve outcomes. Future research should focus on integrating nutritional interventions with dermatological care to enhance recovery and prevent recurrence.
Keyword: Nutritional deficiencies, skin diseases, malnutrition, pediatric dermatology, vitamins, minerals

ABSTRAK

Latar Belakang: Defisiensi nutrisi merupakan masalah kesehatan global yang signifikan, terutama pada anak-anak, karena dapat mengganggu pertumbuhan dan perkembangan. Kulit sering kali mencerminkan tanda-tanda awal defisiensi nutrisi, termasuk ketidakseimbangan makronutrien dan mikronutrien, yang dapat muncul sebagai gejala dermatologis spesifik. Identifikasi dini manifestasi ini sangat penting untuk mencegah morbiditas dan mortalitas jangka panjang.
Tujuan: Tinjauan ini bertujuan untuk mengetahui hubungan antara defisiensi nutrisi dan penyakit kulit pada anak-anak, dengan melihat gambaran klinis, pendekatan diagnostik, dan strategi pengobatan yang efektif.
Metode: Tinjauan literatur komprehensif dilakukan menggunakan studi dan laporan kasus yang relevan mengenai manifestasi dermatologis pada anak akibat defisiensi makronutrien dan mikronutrien.
Diskusi: Defisiensi nutrisi, termasuk malnutrisi energi-protein, defisiensi asam lemak esensial, serta defisiensi vitamin A, B-kompleks, C, dan K, serta zinc dan tembaga, menunjukkan berbagai manifestasi kulit seperti xerosis, dermatitis, hiperkeratosis, hiperpigmentasi, dan petechiae. Diagnosis melibatkan kombinasi riwayat diet, pemeriksaan klinis, dan tes laboratorium untuk menilai status nutrisi. Pengobatan memerlukan suplementasi nutrisi yang disesuaikan dengan defisiensi spesifik, disertai perawatan dermatologis suportif.
Kesimpulan: Manifestasi dermatologis sering kali menjadi indikator pertama defisiensi nutrisi pada anak-anak. Pengakuan dini dan pendekatan multidisiplin

untuk diagnosis dan pengobatan sangat penting untuk meningkatkan hasil. Penelitian di masa depan perlu berfokus pada integrasi intervensi nutrisi dengan perawatan dermatologis untuk meningkatkan pemulihan dan mencegah kekambuhan.
Keyword: Defisiensi nutrisi, penyakit kulit, malnutrisi, dermatologi anak, vitamin, mineral

1. Introduction

Nutritional deficiencies are major contributors to global morbidity and mortality, especially among children.[1,2] Proper growth and development require sufficient intake of macronutrients, including carbohydrates, proteins, and fats, along with essential micronutrients such as vitamins and minerals.[2,3,4] Malnutrition impacts more than 2 billion individuals worldwide, with children being the most at risk. In 2017, approximately 150 million children under the age of five were affected by malnutrition, resulting in an estimated five million deaths annually.[2]

The skin, as a visible organ, often reveals changes caused by nutritional deficiencies.[1] Both micronutrient and macronutrient imbalances can manifest as skin abnormalities. Micronutrients, including vitamins and minerals, are essential in small amounts, and their deficiency can disrupt critical processes such as collagen synthesis, skin barrier maintenance, and immune function.[2] Similarly, macronutrient deficiencies, such as insufficient protein or essential fatty acids, can impair wound healing, alter skin hydration, and weaken the skin's structural integrity.[2] These disturbances highlight the skin's role as a reflection of overall nutritional health.[2] Manifestations such as dermatitis, pigmentation abnormalities, and xerosis are common, but overlap in clinical presentations makes timely diagnosis challenging.[1,2] Identifying the root cause of these deficiencies involves a comprehensive evaluation that integrates dietary history, physical examination, and laboratory tests. Addressing malnutrition effectively requires early diagnosis and targeted therapy to reduce long-term morbidity and mortality.[2] This review article aims to explore the dermatological manifestations of malnutrition, emphasizing the clinical features, diagnostic approaches, and management strategies to improve outcomes in affected individuals.

2. Method

Literature This review article was developed by gathering information from scientific journals, dermatology textbooks, and case reports focused on nutritional deficiencies and their dermatological manifestations in children. Sources were selected based on relevance and credibility, highlighting studies that addressed the clinical features, diagnostic methods, and treatment approaches for these conditions. The literature search included both recent findings and foundational references to provide a comprehensive overview of the topic. Keywords included combinations such as nutritional deficiency, dermatological manifestations, children, and pediatric nutrition. The search focused on articles published within 2012–2023 to ensure up-to-date insights, while also including seminal studies beyond this range for context. Filters were applied to prioritize peer-reviewed journal articles, case reports, and clinical studies in English.

3. Result and Discussion

Nutritional deficiencies significantly impact children's health, with skin manifestations often serving as early and visible initial manifestations.[3] Malnutrition affects all organ systems, as energy and nutrients are essential for sustaining biochemical and physiological processes. Deficiencies in both macronutrients and micronutrients disrupt these processes, leading to impaired physiological functions and systemic health disturbances.[4] Nutritional deficiencies can impair key cellular processes such as keratinocyte proliferation, differentiation, and lipid synthesis, as well as disrupt immune responses and antioxidant defenses.[5]

Adequate micronutrient levels are essential for maintaining the integrity and functionality of the body’s physical barriers, immune cells, and overall immune defense mechanisms. Emerging evidence suggests that the daily micronutrient requirements needed to optimize immune responses may exceed current recommended dietary allowances.[5] The immune system operates as a sophisticated defense network, employing physical and biochemical barriers, specialized immune cells, and antigen-specific antibodies to combat pathogenic microorganisms. Each phase of this immune response, from barrier protection to the activation and regulation of innate and adaptive immune processes, is critically dependent on the availability of specific micronutrients to ensure optimal functionality.[5]

Cutaneous manifestations are key indicators of severe acute malnutrition, with multifactorial causes that are challenging to describe purely in dermatological terms. The severity of skin lesions can predict morbidity and mortality, and specific deficiencies. Early recognition of these manifestations is critical, as nutritional deficiencies often occur in conjunction with other systemic symptoms, complicating diagnosis and management.[3,4,6] Proper examination, investigation, and classification of these lesions can enhance patient outcomes.[4] Below is a detailed discussion of the most common nutritional deficiencies and their associated dermatological presentations.

1. PROTEIN ENERGY MALNUTRITION

Protein-energy malnutrition, often referred to as severe acute malnutrition, is the most prevalent form of nutritional disorder globally.[1]"The protein energy malnutrition subtype is defined based on deficiencies in protein and total caloric intake, resulting in body weight and impaired development and physiological changes.[1,2] Marasmus and kwashiorkor represent the two primary types of acquired protein-energy malnutrition. Marasmus occurs due to a lack of both protein and calories, whereas kwashiorkor arises from a prolonged protein deficiency despite sufficient calorie consumption.[1,2,3]

Marasmus

Marasmus is a failure due to inadequate calorie and protein intake. Weight is significantly reduced relative to height, resulting in extreme thinness with a body weight less than 60% of the expected for age. In advanced stages of marasmus, children exhibit vital signs such as lowered body temperature and bradycardia.[7] The skin manifestations in marasmus are indeed varied. Common findings include thin, wrinkled skin that appears finely scaly, often accompanied by edema. The loss of buccal fat pads, leading to a characteristic "monkey facies" or aged appearance, is frequently observed. Lanugo hair, a compensatory mechanism for reduced subcutaneous fat, may also develop. Hair changes, such as thinning and fragility, are common, while nails often become thin and prone to cracking. Mucocutaneous manifestations, including angular stomatitis, a pale tongue, and mucosal atrophy, are also frequently seen. Less typical but noteworthy manifestations include erythema, erythroderma, petechiae, ecchymoses, scaling, and purpura.[1,2]

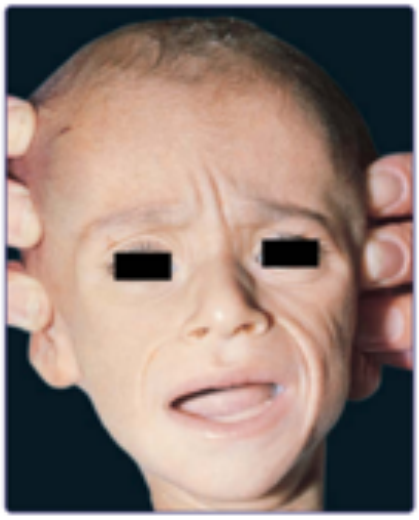

Figure 1. Marasmus “monkey facies” Wrinkles appear on the skin and subcutaneous fat decreases.[2]

Kwashiorkor

Kwashiorkor is a type of protein-calorie malnutrition characterized by generalized edema and an increase in body weight exceeding 60% of the expected weight for age.[8] This condition is commonly observed in children between the ages of 6 months and 5 years.[2,8]

Skin changes associated with kwashiorkor include hyperpigmented areas and cracking, often resembling "enamel paint" or "crazy plaster" in pressure-prone regions. Erythema, erythroderma, and desquamation are also frequently observed.[1] In kwashiorkor, skin changes evolve over several days, beginning with dryness and atrophy and advancing to diffuse hyperkeratosis and hyperpigmentation. A characteristic feature is the shiny, varnished appearance of hyperpigmented areas. Hair typically appears sparse, dry, and brittle, often displaying a reddish-yellow tint. The "flag sign," marked by alternating bands of normal and pale-colored

hair, indicates fluctuations between adequate and inadequate nutrition. Nail abnormalities are also common, including thinning, fissures, ridges, or pronounced koilonychia.[1,9,10]

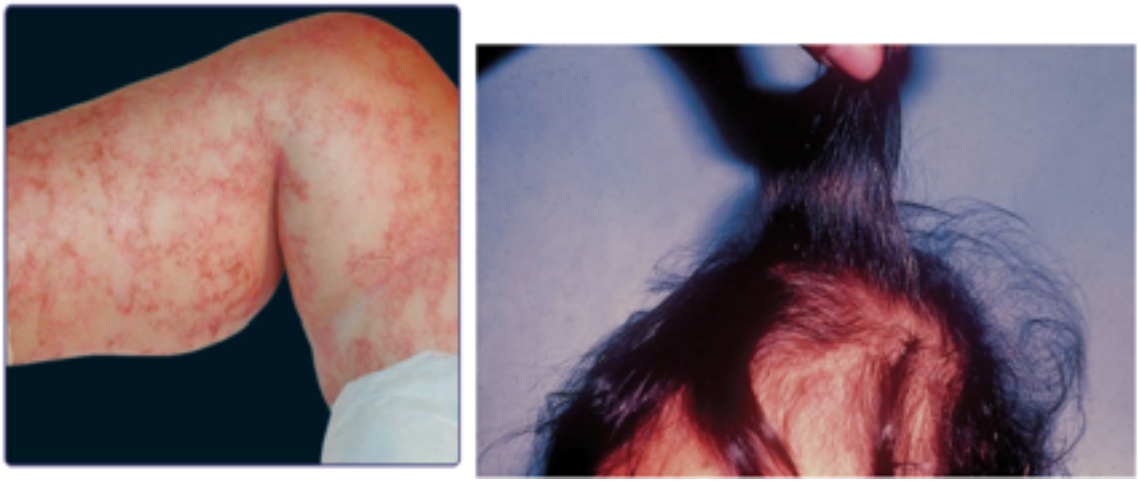

Figure 2. A. "Enamel paint or crazy pavement" kwashiorkor dermatitis. **B.** *Flag sign*.[1]

Management

A multidisciplinary approach and hospitalization are frequently necessary for patients with severe acute malnutrition because of the potential for hypoglycemia, hypothermia, dehydration, and sepsis. When a patient is admitted with suspected sepsis, empiric antibiotic therapy may be considered, and any infection that is found should be properly treated.[2] Research by Ubesie et al. recommends exclusive breastfeeding for the first six months of life and continued breastfeeding until the age of two, as protein-energy malnutrition is most prevalent in children aged 6 to 24 months.[11] Skin lesions associated with malnutrition typically resolve once the child receives adequate nutrition.[1]

2. FATTY ACID DEFICIENCY

Essential fatty acids, including linoleic acid, linolenic acid, and arachidonic acid, play critical roles in various functions, such as the formation of lamellar granules in the skin.[1] Deficiency in these fatty acids can lead to skin symptoms like dryness, scaling, rough texture, erythema, interstitial erosion, and hair loss (alopecia).[12] Systemic effects may include fatty liver infiltration, heightened vulnerability to infections, weakened immune response, anemia, thrombocytopenia, and impaired growth.[1,2,3] Laboratory findings typically reveal reduced levels of linoleic and arachidonic acids, along with elevated plasma levels of eicosatrienoic acids (5, 8, 11).[2]

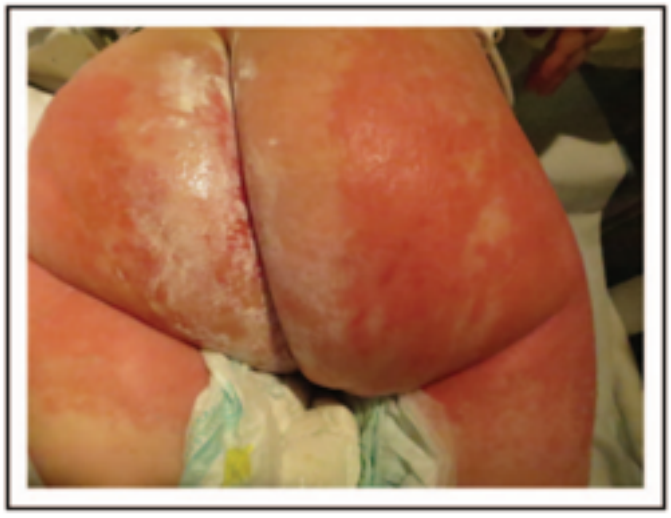

Figure 3. Skin dryness, scaling, rough texture, erythema, interstitial erosion; manifestations of essential fatty acid deficiency.[3]

Management

The course of treatment involves substituting essential fatty acids. The severity of any concomitant dietary deficits will determine the prognosis. Applying sunflower seed oil or safflower oil, which are high in linoleic acid, can help alleviate skin lesions caused by fatty acid deficiency; nevertheless, oral or intravenous essential fatty acid supplementation is typically the preferred course of treatment. To prevent essential fatty acid deficiency, 1% to 2% of daily calorie intake should be derived from essential fatty acids.[2,13]

1. VITAMIN DEFICIENCIES

Vitamins are organic compounds that, in small quantities, serve as catalysts for cellular metabolic processes vital for the proper functioning and growth of tissues. Both excessive and insufficient levels of vitamins can lead to alterations in the skin and mucous membranes. Over 2 billion people worldwide are affected by deficiencies in vitamins and minerals, with iron, vitamin A, and zinc deficiencies being particularly common, specially pregnant women and children younger than 5.[1,14,15]

Vitamin A Deficiency

Vitamin A is a fat-soluble vitamin found in high concentrations in milk, liver, and animal fats.[1,16] Provitamin A, carotenoids, and plant-based foods like vegetables and fruits are the primary sources of vitamin A, with 6 µg of carotene being equivalent to 1 µg of retinol. The daily requirement for children ranges from 500 to 1500 µg.[1] Vitamin A plays a crucial role in the functioning of the eyes, immune system, and skin. It promotes early inflammatory responses, collagen production, and angiogenesis. Its most significant function in the skin is in cornification and keratinization.[1]

Phrynoderma, a condition resulting from vitamin A deficiency, is marked by follicular papules with central keratotic plugs, typically located on the extensor surfaces of the limbs. The hair becomes thin and brittle, and the skin appears dry and rough overall. Common skin symptoms include erythema, skin fragility, desquamation, dry mucous membranes, and widespread alopecia.[1,16]

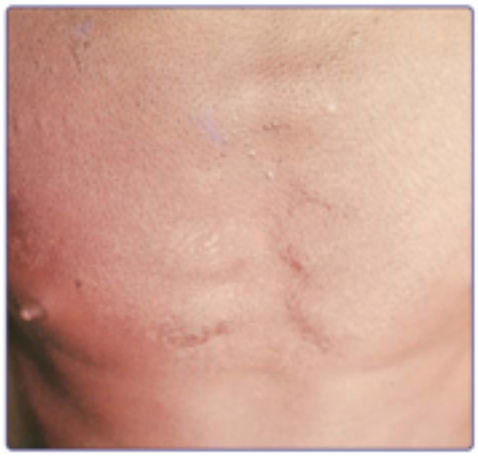

Figure 4. Perifollicular hyperkeratosis of the thoracic region[2]

Management

The suggested daily dosage of vitamin A for treating deficiency ranges from 600 to 3000 mcg, continuing until the symptoms subside and serum levels return to normal.[2] Follicular papules can be treated with keratolytic such as salicylic acid, or topical retinoids. Improvement in the skin occurs slowly over several weeks or months.[8]

Vitamin B Deficiency

Vitamin B is a water-soluble nutrient essential for humans to prevent metabolic disturbances. The B vitamin group includes riboflavin (vitamin B2), niacin or niacinamide (vitamin B3), pyridoxine (vitamin B6), and cobalamin (vitamin B12).[1]

Vitamin B2 Deficiency

Vitamin B2, also known as riboflavin, is a water-soluble vitamin present in various animal products, green leafy vegetables, and yeast. Clinical signs of riboflavin deficiency include erythema, necrolysis, and mucositis. Chronic riboflavin deficiency, or ariboflavinosis, typically develops 3 to 5 months after a diet

lacking sufficient riboflavin. The condition often begins with angular stomatitis, characterized by small papules at the corners of the mouth that grow larger, leading to significant maceration and frequent bleeding.[1,2]

Riboflavin deficiency can be diagnosed through screening tests, such as measuring erythrocyte glutathione reductase activity. Administering riboflavin supplements is an effective way to confirm the deficiency. Laboratory tests may reveal normochromic normocytic anemia. The recommended daily intake of riboflavin is 0.6 mg per 1000 kcal. For infants and children, the daily dosage is 1 to 3 mg, while for adults, it is 10 to 20 mg per day.[2]

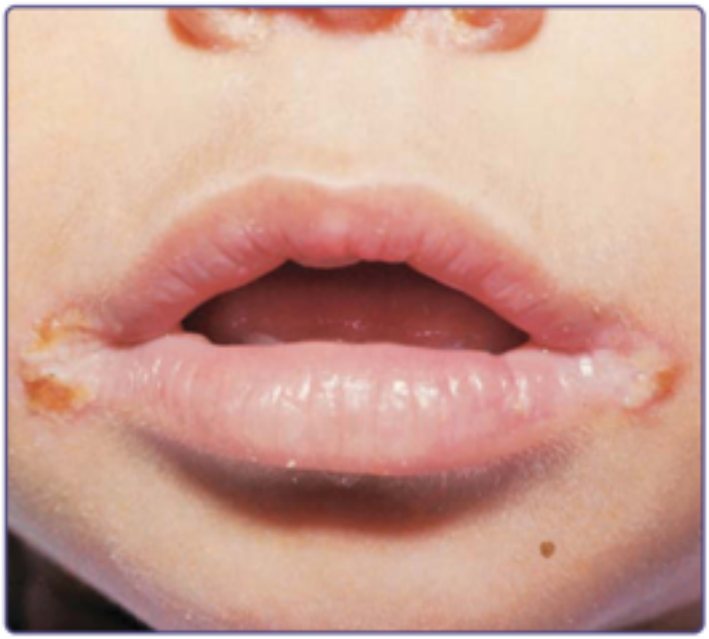

Figure 5. Riboflavin deficiency, angular stomatitis[2]

Vitamin B3 Deficiency

Vitamin B3, also known as niacin, is a water-soluble essential nutrient that plays a key role in the production of nicotinamide adenine dinucleotide (NAD). Niacin is obtained from food sources like grains, meat, milk, and nuts, or it can be synthesized within the body from tryptophan.[2,7]

Pellagra is a hallmark clinical sign of niacin deficiency, typically presenting with dermatitis (often painful rather than itchy), diarrhea, and dementia. Skin symptoms include symmetrical erythema in sun-exposed areas such as the hands, neck, and face.[2,7] These skin changes can appear suddenly and worsen with sun exposure. Lesions on the hands are referred to as "gloves pellagrous," while those around the neck are called "Casal's necklace." The skin thickens, peels, and may develop pigmentation, and in some cases, blisters may form. Mucosal involvement can lead to cheilitis, atrophic glossitis, and inflammation and pain in the perianal and vaginal areas.[8]

Figure 6. A. Pellagra. Casal's necklace. **B.** pellagrous gloves.[2]

Management
Treatment typically involves administering 500 mg of nicotinamide or nicotinic acid daily for several weeks. Skin lesions usually take about 3 to 4 weeks to resolve.[2]

Vitamin B6 Deficiency
Vitamin B6, also known as pyridoxine, is commonly found in both plant and animal sources, including meat, wheat, and milk. The daily requirement ranges from 10 to 20 mg. Pyridoxine is involved in the metabolism of essential fatty acids and amino acids. Deficiency in vitamin B6 is often linked to medications like isoniazid, but it typically occurs alongside other nutrient deficiencies. Clinical signs may include seborrheic dermatitis affecting areas such as the head, neck, shoulders, buttocks, and perineum. The clinical picture usually overlaps with niacin deficiency, namely photodermatitis, glossitis, andcheilitis.[1,2]

Laboratory examination revealed low levels of plasmapyridoxal-5-phosphate. Pyridoxine dosage should be adjusted according to age and gender. Adult men require 2 mg per day, adult women need 1.6 mg per day, and infants should receive 0.3 mg per day. Oral lesions typically improve within a few days, skin lesions and hematologic changes clear up within a few weeks, and neurological symptoms may take several months to resolve.[2]

Vitamin B12 Deficiency
Vitamin B12 (cyanocobalamin) deficiency can result from insufficient intake, impaired secretion, or inhibition of the intrinsic factor in the stomach, as well as abnormalities in the receptor sites of the ileum. The daily requirement ranges from 1 to 5 µg. Early detection of vitamin B12 deficiency is crucial to avoid potential complications in brain development.[1,17]

The most frequent skin manifestation is hyperpigmentation. Predilection for hyperpigmentation is the area of the dorsum of the manus and pedis, especially at the interphalangeal joints, which is called 'knuckle pigmentation' and can spread to wider areas of the body. Hyperpigmentation can give different patterns including a reticular appearance, 'honeycomb', or in the form of livedo reticularis. This pattern is generally homogeneous, persistent, and dark brown. Hair and scalp abnormalities can be found in the form of brownish areas on the scalp and thin and sparse hair.[18] Laboratory examination showed a decrease in serum cobalamin levels (200 pg/mL) as well as a picture of macrocytic anemia with increasing numbers mean corpuscular volume (MCV).[19]

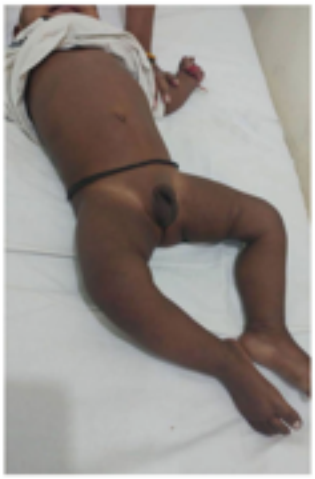

Figure 7. Generalized, uniform, or homogeneous hyperpigmentation of the skin[18]

Management

Treatment for the acute phase of vitamin B12 deficiency involves intramuscular injections of 500 µg of vitamin B12 for 3 to 5 days, followed by oral supplementation of 15–30 µg per day for a period of 6 to 12 months. Dermatological improvement is gradual. Hyperpigmentation gradually disappears one week to 4 months after therapy. Complete resolution takes several years.[2,7]

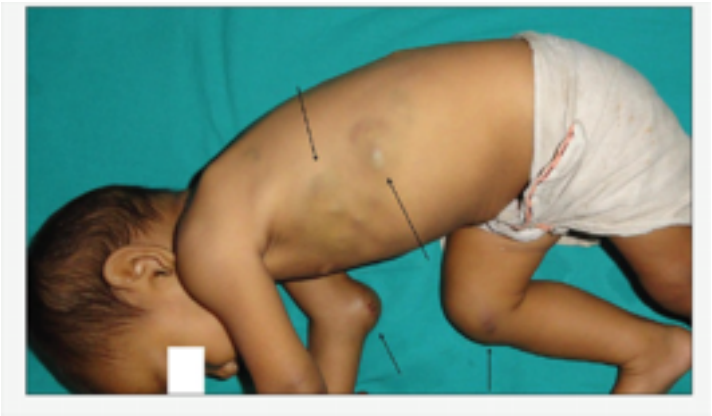

Figure 9. Bleeding on the skin multiple nodular lesions[21]

1. MINERAL DEFICIENCIES

Copper Deficiency

"An essential component of many enzymatic and metabolic activities is copper. Fish, oysters, wheat, cattle, pork liver, eggs, and raisins are foods high in copper."[1,2] Menkes syndrome is a manifestation of congenital copper deficiency, caused by a recessive X-linked mutation in the copper-transporting P-type adenosine triphosphatase, which is expressed in almost all tissues, except the liver.[2] This condition is characterized by hypopigmentation and neurological degeneration.[2] Infants with Menkes syndrome initially appear normal, but by two to three months of age, they begin showing signs of lethargy, hypothermia, drowsiness, hypotonia, seizures, failure to thrive, intellectual disability, and bone abnormalities. The patient typically has a normal facial appearance, pale skin, and sparse, brittle, curly hair.[2,22]

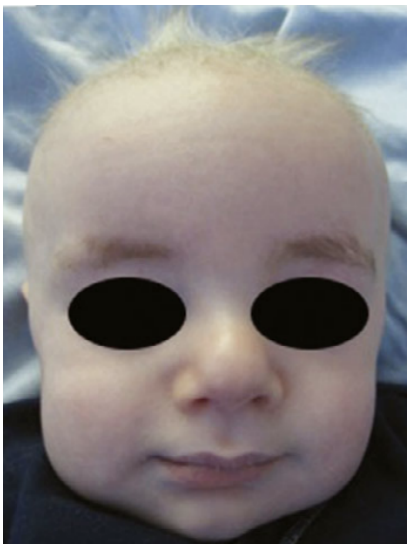

Figure 10. Sparse hair and hypopigmentation in Menkes syndrome[22]

Zinc Deficiency

" Zinc is predominantly found in meat, dairy products, nuts, seeds, liver, fish, and oysters.[3] The recommended daily intake is 3–5 mg for infants and 5–10 mg for children.[2]

Inherited zinc deficiency, known as acrodermatitis enteropathica (AE), is caused by mutations in the ZIP4 gene, leading to impaired zinc absorption in the small intestine. Another form, transient neonatal zinc deficiency, results from mutations in the zinc transporter gene ZnT2, causing low zinc levels in breast milk. Both conditions are typically accompanied by symptoms such as dermatitis, diarrhea, and alopecia.[7,23] Skin lesions often appear early in life, after weaning, and include erythematous plaques around the lips, tartar, and erosions. Affected individuals may also have fine, sparse hair and dystrophic nails, with common findings of blepharoconjunctivitis and cheilitis. Children with these conditions are often apathetic, irritable, and experience failure to thrive.[1]

Initial manifestations are usually visible angular cheilitis and paronychia. The rash starts as scaly, eczematous plaques that evolve into vesicular, erosive lesions with crusted edges, particularly around the mouth, perineum, limbs, and scalp. In milder cases or chronic deficiency, psoriasiform dermatitis may also develop on the hands, feet, and knees.[19]

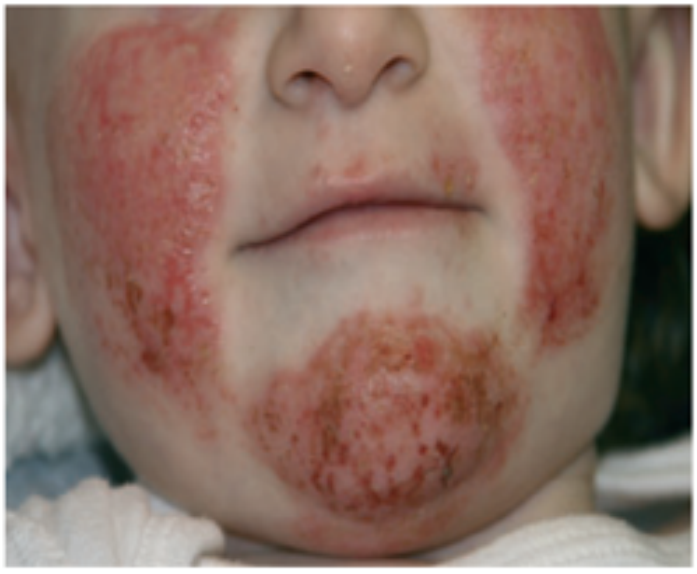

Figure 11. Skin manifestations of zinc deficiency like *acrodermatitis enteropathica*[7]

The definitive method for diagnosing zinc deficiency is through low plasma zinc levels, with the normal range being 70–250 mcg/dL. Serum alkaline phosphatase testing serves as a rapid indicator of zinc status; typically, the levels may be normal or low, but taking zinc supplements will result in an increase in serum alkaline phosphatase, which helps confirm the diagnosis.[2]

Management

Administering zinc supplementation enterally or parenterally."Initial improvement is frequently noticed in a few days, with a typically rapid clinical response. First, complains and irritability go away, then the skin lesions become better.[2]"While there are a number of zinc formulations on the market, zinc sulfate is the most often utilized enteral formulation. It is advised to use parenteral zinc chloride supplements. For mild to moderate zinc deficiency in children, one to two daily doses of 0.5 to 1.0 mg/kg zinc are advised. In cases of intestinal malabsorption-related zinc insufficiency, higher doses could be required. Monitoring serum zinc levels is necessary while on medication. Treatment for AE patients must last a lifetime. The required amount of zinc supplements for patients with zinc deficiencies may vary depending on the underlying cause. It is important to note that excessive zinc levels can interfere with copper metabolism.[2]

4. Conclusion

Various cases of nutritional deficiencies may present with similar clinical features, making it essential to determine the underlying cause through a thorough history-taking, including dietary intake, dermatological assessment, and supporting investigations. Early diagnosis of skin manifestations is crucial to ensure effective treatment and achieve favorable outcomes with an improved prognosis.

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