Cutaneous tuberculosis can be caused by consuming cow milk contaminated with Mycobacterium bovis or by droplet infection with M tuberculosis. The correct diagnosis is often significantly delayed because CTB is not routinely considered in the differential diagnosis or because investigations fail to reveal the presence of M tuberculosis.4

Our case illustrates that scrofuloderma, though a rare disease in industrialized countries, should still be considered in the differential diagnosis of unusual abscesses and nodules of the neck. Skin testing and interferon-γ release assay can support the clinical diagnosis. Since PCR has been shown to have a limited sensitivity and specificity (eg, 88% sensitivity and 83% specificity5), there is a risk of failure to detect mycobacteria in skin samples by relying solely on PCR. Therefore, PCR should always be accompanied by culture.6

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Dermatitis and Dangerous Diets: A Case of Kwashiorkor

Although uncommon, kwashiorkor continues to occur in developed nations. A recent case highlights the fact that such occurrences are typically the result of well-meaning dietary restriction in the setting of nutritional ignorance. Telltale skin and hair changes should prompt a thorough dietary history and appropriate dietary intervention.

Report of a Case | A young boy presented with a 1-year history of progressive skin, hair, and nail changes after institution of a low-protein diet recommended by an outside physician as therapy for his nonketotic hyperglycinemia (NKH). Examination revealed generalized hypopigmentation with numerous erthematous and denuded patches over his trunk, arms, and legs. Desquamation in a flaking or “paint-chip” pattern was prominent on the upper and lower extremities (Figure 1). His abdomen was distended with dependent edema over the scaprum and extremities. His hair was pale yellow and brittle with patches of alopeica. Fingernails were thin and brittle with distal nail plate splitting.

Laboratory levels were measured as follows: total protein, 5.4 g/dL (normal, 5.7-8.2 g/dL); albumin, 2.8 g/dL (normal, 3.2-4.8 g/dL); and prealbumin, 8 mg/dL (normal, 10-40 g/dL). Aspartate transaminase and alanine transaminase levels were elevated at 76 U/L (normal, <11-34 U/L) and 55 U/L (normal, 10-49 U/L), respectively. (To convert total protein and albumin to grams per liter, multiply by 10; to convert prealbumin to milligrams per liter, multiply by 10; to convert aspartate transaminase and alanine transaminase to microkatal per liter, multiply by 0.0167.) Levels of alkaline phosphatase, total bilirubin, iron, phosphorous, magnesium, and stool alpha-1 antitrypsin were within normal limits. Values for zinc, vitamins A, K, and E and 1,25-vitamin D were above or within reference ranges.

Our patient’s clinical and laboratory findings were consistent with kwashiorkor1 secondary to dietary protein restriction intended as therapy for NKH, a rare disease of glycine metabolism causing accumulation of glycine in the cerebrospinal fluid and leading to subsequent N-methyl-D-aspartate receptor excitotoxic effects or overstimulation of glutamate receptors in the central nervous system. These excitotoxic effects manifest clinically as intractable seizures, severe mental retardation, and permanent neurologic disease.2 Glycine is a nonessential amino acid produced via numerous catabolic pathways; therefore, dietary restriction of glycine has no therapeutic effect on NKH.3 Our patient’s skin changes improved rapidly with increased dietary protein. Figure 2 demonstrates resolution of desquamation and erosions at 1-month follow-up.
Discussion | This case recalls an important theme in recent literature: food fads and unconventional dietary changes may cause iatrogenic nutritional deficiencies, and skin changes are often key signs for diagnosis. In a 2010 review of pediatric kwashiorkor in the developed world,1 most cases were attributed to nutritional ignorance on the part of well-meaning but misinformed parents who had substituted lower-protein food in an effort to alleviate perceived or actual food allergies and intolerances. A diagnosis of atopic dermatitis was a common theme: nutritional deficiency–related skin changes are often initially misdiagnosed as atopic dermatitis, and atopic dermatitis frequently triggers dietary restrictions by parents.1 Similarly, a 2011 article4 reported kwashiorkor in 3 infants with cow’s milk allergy whose parents misguidedly substituted a lower-protein rice milk beverage instead of the nutritionally sufficient hydrolyzed rice protein formula recommended by health care providers. Cases of kwashiorkor in addition to ours have occurred secondary to dietary changes made by health care providers. Two published cases report kwashiorkor developing as an adverse effect of a protein-restricted diet used as treatment for glutaric acidemia type 1.5,6

Well-intentioned food restrictions can result in severe consequences. For the dermatologist, these cases highlight the importance of obtaining a nutritional history, especially in patients with chronic or persistent skin disease. Health care providers should be aware of food fads, cultural practices, and dietary trends as well as current literature relating to nutrition and skin disease. Assessing parents’ and health care providers’ understanding of a child’s nutritional needs, the influence of diet on skin disease, and the nutritional value of popular foods is critical in preventing potentially serious adverse outcomes such as kwashiorkor.

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